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THE MEDICAL CLINICS
OF
NORTH AMERICA

Volume 7 Number 6

CLINIC OF DR. C. F. MOFFATT

ROYAL VICTORIA HOSPITAL

THE ELECTROCARDIOGRAM IN PROGNOSIS

IT no doubt has been the experience of every practitioner to treat certain cardiac cases which frequently, on very insufficient clinical data, have been classified as "myocarditis." This term "myocarditis" has come to be used very loosely to cover various types of circulatory disease, in many instances to cover our ignorance, in much the same manner as we use that much abused and little understood term "auto-intoxication." With symptoms referred to the circulatory system in general and the heart in particular, but, in reality, produced by involvement of another system—not the circulatory—we are sometimes surprised that our patients do not respond to treatment and do not fulfil the perhaps adverse prognosis given.

It is in consideration of this difficulty that I wish to draw your attention to the undoubted assistance which the electrocardiogram can give us in just such problems.

I wish to emphasize, however, that such aid as this method provides must be taken in close conjunction with the usual other clinical examinations; it must supplement, not replace. Too often more is expected of the graphic method than it is reasonable to hope from any one clinical examination. Medicine is not yet an exact science, and if our laboratory methods do not always fall in line with the clinical findings and they, in turn, with the pathologic results, due allowance must be made.

Based on numerous experimental data and checked up by a wide clinical experience, we have come to regard as

normal certain electrocardiograms within specified limits of variation.

An example of a normal electrocardiogram is shown in Fig. 287.

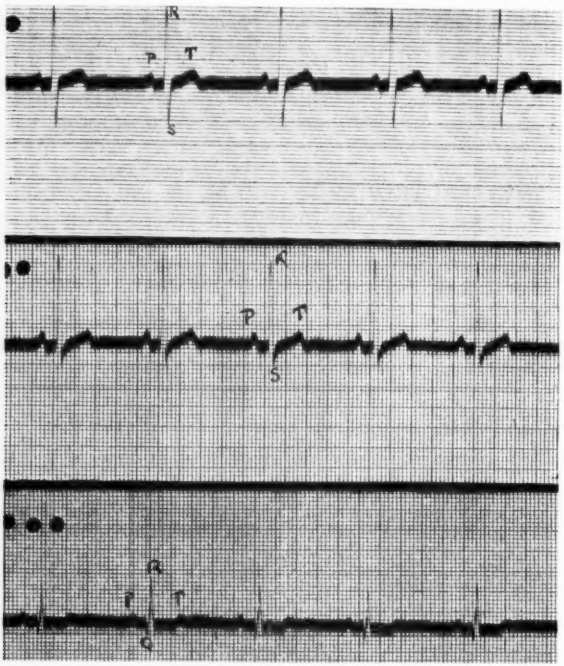

Fig. 287.—A normal electrocardiogram. P represents auricular, Q, R, S, and T, ventricular, activity. In this and subsequent tracings 1 cm. is equal to 1 millivolt; time marker in fifths and twenty-fifths of seconds.

An electrocardiographic tracing does not coincide exactly with the actual muscular contraction of the heart, but is the resultant of the electric changes which take place during muscular activity and, as a matter of fact, precedes the actual contraction by 0.009 to 0.003 second.[1]

[1] Thomas Lewis, The Mechanism and Graphic Registration of the Heart Beat.

The wave marked "P" corresponds to auricular contraction, the complex Q. R. S. and T represent ventricular activity. It is particularly in reference to the wave "T" that we wish to draw certain conclusions.

This terminal ventricular deflection normally is upright in at least the first and second leads; its inversion in the third lead is not of such importance; in fact, such a change is frequently associated with perfectly normal hearts. There are certain conditions which induce temporary change in the direction of the "T" wave.

Chief among such exceptions are those changes evidenced by inversion of this wave, produced by digitalis, and by the application of cold to the apical region of the heart—produced experimentally by drinking large draughts of ice-cold fluid. These changes are of a temporary nature and we are not concerned with them at present. The more permanent alterations of this deflection, however, are important and bear an important relationship to intrinsic changes in the heart muscle, and as such are of great aid in arriving at a more accurate diagnosis and prognosis.

There are other features about the electrocardiographic tracing which are of considerable value in arriving at a more definite idea of muscular function, *e. g.*, conduction within the heart itself. These other factors do not enter into the present discussion, however.

As illustrative of the above points I wish to describe in more or less detail 3 cases of a distinctive type. They are chosen from a series of 63 similar cases which have occurred in the cardiac clinic of the Royal Victoria Hospital during the past two years.

These 3 cases are chosen because they show typical tracings without being influenced by the effect of digitalis.

Case I.—J. W. G., aged fifty-three years, a printer by trade, came under my observation February 7, 1923, complaining of shortness of breath and precordial distress—not pain—on slight exertion. He had been so troubled for the past year or

so, but had continued at his work. Within four to five months previous to my seeing him these symptoms had increased in severity and had been more easily induced. After a long day's work he noticed slight swelling of his ankles. Occasionally he complained of a pounding in his head associated with vertigo. He had no nocturnal dyspnea and there was no orthopnea. His previous health had always been good. His habits had always been temperate; he denied venereal disease. His father died suddenly of heart disease at the age of seventy-nine; his mother at the age of eighty-two died of acute dilatation of the heart.

On examination he was a well-built man somewhat overweight and of pale complexion. He appeared older than his stated age. He was quite comfortable while quiet in bed, but the exertion of getting out of bed and walking about the ward produced slight dyspnea and a rise in pulse-rate. The circulatory system was of interest. The peripheral circulation was fairly well maintained; there was no edema, no cyanosis. The peripheral arteries were moderately sclerosed, the rate averaged 100 per minute, a regular rhythm was occasionally interrupted by extrasystoles, the volume was good, the blood-pressure on admission was systolic 210, falling in a few days to 180, the diastolic remained stationery at 95 mm. of Hg. Between the systolic pressures of 190 and 180 there was a definite "pulsus alternans," *i. e.*, half the beats coming through at 190 systolic and all appearing when the pressure was lowered to 180. This feature persisted while under observation. The heart was enlarged to percussion: 16 cm. in its total transverse diameter, but mainly to the left. The width of the vessels at the base of the heart was not enlarged; this was corroborated by an *x*-ray examination. The heart sounds were clear, the aortic second sound being accentuated. There was a transient systolic apical murmur regarded as of relative mitral insufficiency.

The urinary findings indicated a chronic interstitial nephritis.

The blood Wassermann was negative.

An electrocardiogram taken February, 10, 1923 (Fig. 288),

showed a relative left-sided hypertrophy; the conduction time was normal. The final ventricular deflection "T" in the first lead was negative and persisted so. He never had had digitalis.

A thorough search for some focus of chronic infection—*e. g.*, infected teeth or sinuses—was negative.

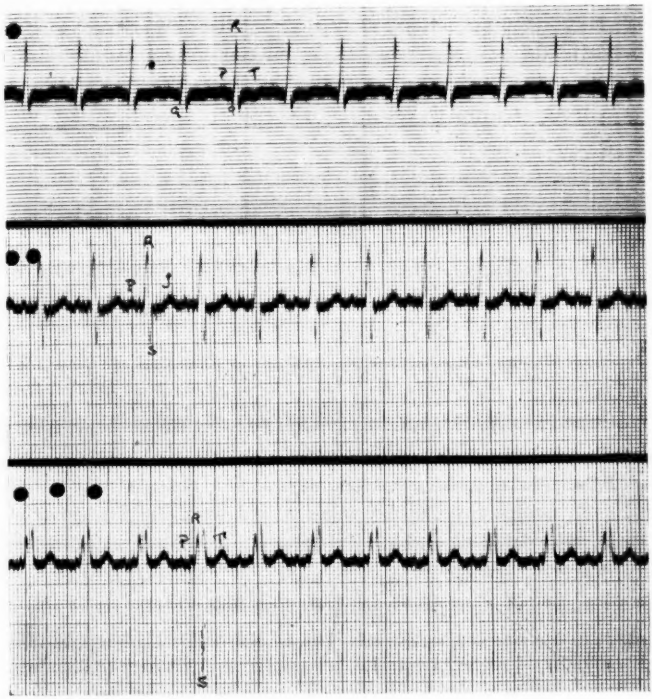

Fig. 288.—Case I. Electrocardiogram showing persistent "T" wave negativity in Lead I; also left-sided preponderance.

He was sent home and advised to take a complete rest.

His own physician reported an improvement for a time; that the dyspnea lessened; that the pulsus alternans persisted; and that the pulse-rate tended to higher rates than formerly on slight exertion. No other change was noted.

In January, 1924, while alighting from a street car, he

dropped dead—within one year after his condition was definitely established.

We might regard this case as one of hypertension where a chronic arteriosclerotic process had involved the whole arterial tree, including, among other organs, the kidneys and heart. A degenerative condition of the arteries not due to any discoverable focus of infection during his lifetime nor to syphilis, as borne out by a negative blood Wassermann test, a condition which seemed to have more or less a selective action upon the coronary arteries with resulting myocardial weakness.

It might be suggested that the combination of symptoms complained of, the great restriction of his field of cardiac response in addition to the pulsus alternans, were sufficient evidence to give a bad prognosis.

I feel, however, that here the result of the electrocardiogram in addition to the above was a very definite confirmatory aid in making our unfavorable prognosis.

No autopsy was obtained.

Case II.—W. K., a man aged fifty-three was admitted to the surgical service, Royal Victoria Hospital, August, 1922, with a cholelithiasis and acute cholecystitis. Ten days previously he had been seized with severe pains in the right hypochondrium, radiating through to the back, associated with nausea, vomiting, and increasing jaundice. These symptoms are mentioned because frequently similar complaints with the exception of the jaundice are found associated with coronary thrombosis and are frequently misinterpreted as gall-bladder disease.

On admission he was perceptibly dyspneic. He gave the information that since January, 1922, he had been troubled with dyspnea on exertion, which formerly he had performed without inconvenience. Beyond a sense of constriction about the chest he never had any severe precordial pain. In January, 1922, he had an attack of so-called "cardiac asthma," obtaining relief through rest and digitalis over a period of six weeks, so that here we have at least from January, 1922, and

most likely before, a beginning of very definite cardiac symptoms.

His previous health had been good. Of infections he had one—typhoid fever at the age of twenty-six years. He had always worked hard; he smoked heavily, but used alcohol sparingly. He was a well-developed and nourished man. The skin was deeply jaundiced, and he suffered considerable pain referred to the gall-bladder region. He showed other signs of obstructive jaundice, acholic stools, containing much undigested fat, high-colored urine, with much bile-pigment. As regards the circulation, there was a certain amount of cyanosis of the finger-tips, the pulse was regular, 76 in rate; the blood-pressure was systolic 140, diastolic 80 mm. of Hg; the peripheral arteries were sclerosed and tortuous; there was a faint capillary pulse, but no marked arterial pulsation.

The heart was enlarged to the left, the vessels at the base were not wider than normal. On auscultation the heart sounds were somewhat distant, there was a definite murmur at the apex of mitral insufficiency, and over the third left intercostal space a faint diastolic murmur of aortic insufficiency was heard.

The lungs were moderately emphysematous and showed no signs of stasis at the bases. An *x*-ray of the heart confirmed the physical findings of an enlarged heart both to the right and the left; there were no signs of aortitis or aneurysm.

He submitted to two major operations, the first, September 20th, when many gall-stones were removed. For three days there was a febrile reaction following operation with pulse-rates ranging as high as 130 to 140 per minute. A few days later a second operation was performed to relieve an inflammatory obstruction. Following the last operation, both anesthetics being gas and oxygen, he did not make such a good recovery, there being considerable dyspnea, palpitation, and tachycardia; the auscultatory phenomena remained unchanged. Convalescence was rapid after the normal flow of bile was re-established.

An electrocardiogram was taken on November 8, 1922, at least four weeks after his last digitalis medication (Fig. 289).

It differed in no respect from one taken on admission, previous to any digitalis medication. In all three leads the final ventricular deflection "T" is inverted. The tracing showed no relative preponderance, one side of the heart over the other, and the conduction time was normal.

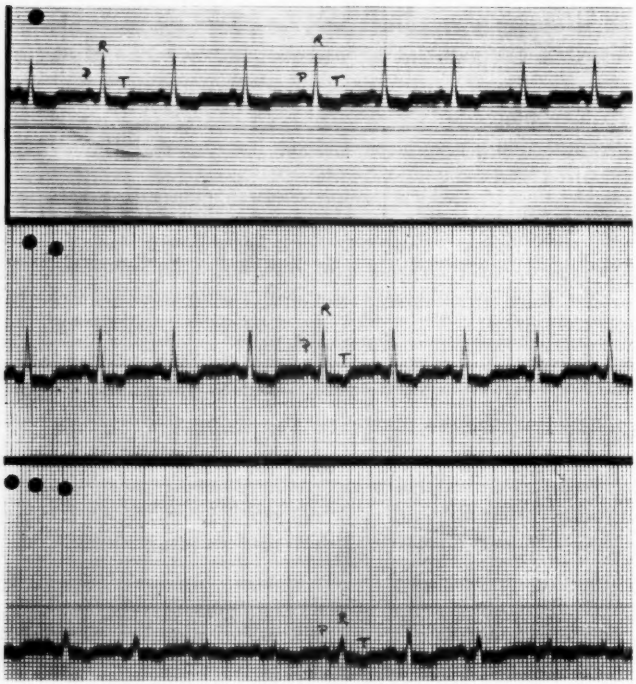

Fig. 289.—Case II. Electrocardiogram showing inversion of the "T" wave in all three leads.

He left the hospital November 22, 1922, in a fair state of health, but, bearing in mind previous experiences, he was advised to convalesce in the South, with strict instructions to rest, and his friends advised as to possibilities. On January 3, 1923, three months after first coming under observation, he dropped dead.

This type of patient differs considerably from that of Case I, "myocarditis," if you wish, but there was definite evidence, in addition, of the endocardium being involved. An aortic endocarditis with insufficiency; the mitral regurgitation was regarded as being of a relative nature due to dilatation of the auriculoventricular ring, and not due to organic mitral disease. Dilatation had been added to hypertrophy and dominated the picture. There had been no definite anginal attacks; he underwent two major operations, with a stormy reaction after the second, but made a good convalescence. He left the hospital in such improved condition that, judging by physical signs only, one might fairly have given a reasonably good prognosis; the distinctive and persistent findings in the electrocardiogram, however, prompted an adverse prognosis.

Case III.—This third case which I wish to speak about, while one of degenerative arterial changes with hypertension, differed considerably in its mode of progression and termination from that of the previous two.

A man sixty-three years of age, of short stocky build, for several years past had known that he was suffering from high blood-pressure, and had taken long vacations from his occupation of broker, in consequence.

I first saw him in June, 1923, when he gave the history that for the past year and a half he had suffered from a "gnawing, clutching, oppressive sensation over the heart," particularly after exercise or after a heavy meal. This sensation never amounted to severe pain and usually responded to rest. For the past nine months he had been short of breath even while walking on the level. At times, however, for periods of days he was entirely free of these complaints. About the beginning of June, 1923, he had an attack of severe nocturnal dyspnea unassociated with severe pain, which lasted several hours; a similar, though less severe, seizure was repeated a few nights later. Temporary relief was obtained by the eructation of gas; vasodilators—amyl nitrite and nitroglycerin—were of no avail in relieving the attacks. For a period of about a week succeed-

ing this he had afternoon rises of temperature—100° to 101° F. He was admitted to the Royal Victoria Hospital a short time after, and during his stay there he was afebrile.

Of somewhat athletic tendencies as a youth, he had been accustomed to use tobacco to a moderate excess, alcohol he always used temperately.

Of infections he had had scarlet fever at the age of eight years, influenza in 1918, and at that time his blood-pressure was recorded as systolic 185 and diastolic 115 mm. of Hg. The main findings of his physical examination were as follows: The peripheral arteries were definitely sclerosed and tortuous, the rate was 90, the rhythm regular, and the blood-pressure was 220 systolic and 140 diastolic. The examination of the heart showed a large outline, dilated mainly to the left 14 cm. from the M. S. L., the dulness over the vessels of the base of the heart was somewhat enlarged. The heart sounds were clear and sharp, the aortic second sound being ringing and accentuated, there were no murmurs. The *x*-ray examination showed a large heart of the sessile type, with a slight widening of the aortic arch, but no evidence of aneurysm.

With rest in bed the symptoms improved, his blood-pressure falling to systolic 190 and diastolic 110 mm. of Hg.

An electrocardiogram taken at this time (Fig. 290) showed a relative left-sided preponderance, and the "T" deflection in Lead I was inverted. The urinalysis revealed a fairly high specific gravity, a constant presence of albumin, and many hyaline and granular casts. The blood chemistry, however, was normal. In the other systems there was nothing of note.

Relieved by his short stay in the hospital and a complete rest, he felt better, and he left the hospital and went home to rest, but by October, 1923, the same symptoms, with increased frequency and severity, had again returned. He returned to hospital in October. An *x*-ray examination of the gastro-intestinal tract, accessory nasal sinuses, and teeth was negative. An electrocardiogram repeated showed essentially the same features as in Fig. 290, the inversion of the "T" wave in Lead I persisting.

From this time on to his death (December 11, 1923), his symptoms became rapidly worse in spite of everything that could be done. Apprehension and precordial distress by day, and marked dyspnea and orthopnea by night, preventing sleep, combined to rapidly use up what little reserve power there was left.

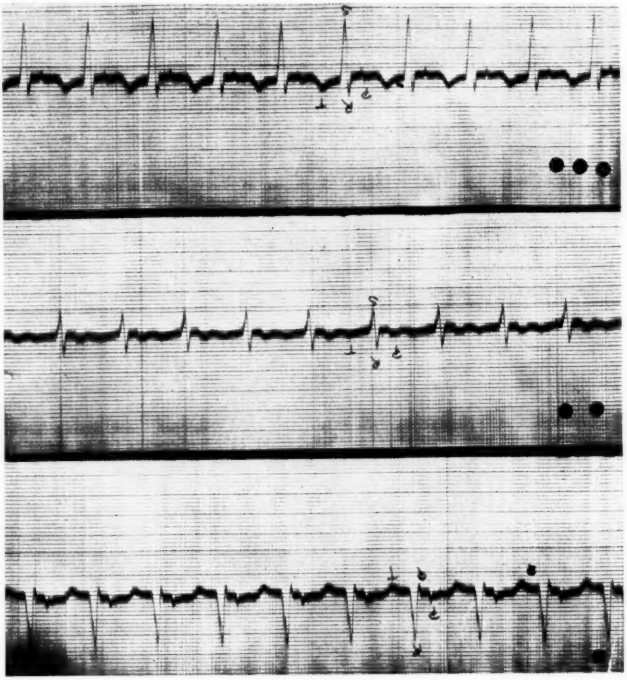

Fig. 290.—Case III. Electrocardiogram showing persistent inversion of the "T" wave in the first lead; a slight prolongation of the auriculoventricular conduction time, and left-sided preponderance.

With all the signs of a complete break in the compensation of his circulation—dyspnea, orthopnea, cyanosis, and general anasarca—he became unconscious and died a typical cardiac death.

An autopsy was permitted, and the findings were as fol-

lows: The heart was large and dilated, particularly on the right side. The myocardium was greatly thickened. There were numerous whitish fibrotic looking streaks in the apical region

Fig. 291.—Case III. A barium injection of the coronary circulation which shows the relative thickness of the right and left ventricular walls; also an area of infarction in the left ventricular muscle toward the apex (marked X).

of the left ventricular wall extending up toward the base for an interval of 9.5 cm.

The endocardium was intact. The abdominal aorta showed patchy sclerosis and numerous calcareous plaques near its

bifurcation. In the other organs were found old infarct scars in both kidneys, lungs, and spleen, and a terminal bronchopneumonia. Histologic examination of the fibrotic area in the left ventricular wall showed a very extensive replacement of the muscle tissue by fibrous scar tissue, well circumscribed. It did not involve the ventricular muscle in its entirety. In this same area the vessel walls showed a marked thickening. Diagnosis: Coronary thrombosis with infarct formation.

An injection of the coronary circulation with barium mixture was made in Professor Oertel's laboratory, and I am indebted to him for permission to use the plate as well as the pathologic findings. The heart is injected through the coronary circulation, an *x*-ray plate is taken, and from it a print is made. The print (Fig. 291) shows the relative thickness of the left and right ventricular walls, the former particularly being much increased in width.

The main point to which I wish to draw attention is the clear area toward the apex, in the left ventricular wall, where apparently the circulation has been cut off. It is the same area which, on sectioning, shows definite coronary thrombosis and replacement fibrosis, due to an infarct.

Looking back upon the development, progress, and termination of Case III, the outstanding facts are, first, hypertension, then gradually a curtailing of his reserve power, with the production of symptoms—dyspnea, precordial distress, etc. The sudden onset of nocturnal dyspnea early in June, 1923, might well have been the result of the coronary thrombosis, with resulting cardiac infarction. Such a complication, throwing out of action a considerable portion of the left ventricle, must for a time at least involve a change of function in that part of the heart until such time as collateral circulation is established.

To a circulatory system already bearing the brunt of hypertension, handicapped with degenerative changes in the arterial tree and consequent loss of elasticity, such an accident to the left ventricle was quite evidently more than it could compensate for, and from this time on his downward progress was rapid.

These 3 cases are chosen from a series of 63 patients showing decided "T" wave negativity in one or more leads, except the third lead, which have been studied in the cardiac department of the Royal Victoria Hospital during the past two years. No case has been included in which there might exist a doubt as to the heart being under digitalis.

Two years is a somewhat short period of time in which to draw final conclusions as to the ratio of mortality. However, aside from this objection, an approximate ratio of mortality for twelve months works out at 31 per cent. of deaths in such cases presenting this abnormal electrocardiogram, this ratio is high, although other observers have reported even higher rates of mortality. Willius, in the Mayo Clinic, reports a rate of 50.4 per cent. mortality in eleven and two-tenths months.

Significant "T" wave negativity is not always associated with manifest clinical evidence of serious cardiac disease, and it is in just such cases that the electrocardiogram is of considerable aid.

From this series we gain the distinct impression that this abnormal feature of the electrocardiogram is more apt to occur in conditions of progressive vascular degeneration involving in general the whole arterial tree, and in particular the coronary circulation, associated sometimes with symptoms of angina pectoris and very frequently found in cardiovascular disease with hypertension.

Corroborative evidence along this line is found in the experimental work of F. M. Smith,[1] who found that ligation of the descending branch of the left coronary artery in dogs was most constantly followed by inversion of the "T" wave. This inversion may not remain so, but may gradually revert back to its normal type. Ligation was not always followed by the death of the animal, the result appearing to depend upon the size of the artery ligated and the ability of the collateral circulation to re-establish itself. These experimental facts are borne out by numerous clinical cases checked up by autopsy.

With these findings in mind, we feel that the injection

[1] Fred. M. Smith, Arch. Int. Med., October, 1923.

method, as used in Case III, offers many possibilities of detecting blocks in the circulatory tree, where routine sectioning would fail.

I am, therefore, of the opinion that in the type of patient showing evidence of degenerative arterial changes, usually at first with hypertension, with symptoms of myocardial insufficiency, with or without anginal attacks, and frequently classified as "myocarditis," the electrocardiogram may be of the greatest assistance in arriving at a more exact diagnosis and prognosis.

CLINIC OF DR. CHARLES F. MARTIN

ROYAL VICTORIA HOSPITAL

SOME TYPES OF PERNICIOUS ANEMIA

I WOULD like to draw to your attention today 3 patients suffering from a grave anemia, and to discuss with you some of the features in the diagnosis and treatment of the so-called "pernicious anemia" and others that are similar in character.

Case I.—The first patient that you see is a woman aged sixty-eight years, fairly well nourished, who, we are told, has been *suffering from a soreness of the tongue* associated with no accountable cause. A careful inspection of her mouth and a thorough examination of the teeth have revealed nothing abnormal, and the tongue itself, as you may see, apart from being very dry and somewhat glossy, shows no evidence of disease.

The pain that is complained of is more or less constant, not very sharp or paroxysmal, but rather of the nature of a soreness located at the tip and the sides of the tongue, but not on the surface or behind.

Six months ago she underwent a very thorough examination, with practically negative results. I will not go into detail, but will merely mention the essential investigations on that occasion:

Apart from some slight general weakness there were no other subjective evidences of disease. The physical examination showed healthy heart and lungs. The urine was normal, as was also the nervous system. Careful examination was made for foci of infection, but none were found. Streptococcic infection as a cause of soreness of the tongue has long been surmised,

and for that reason special efforts were made to look for this cause.

The digestive organs, examined physically, chemically, and by means of *x*-rays, revealed no abnormality. Chemical examination of the blood showed that the uric acid content was that of a healthy person. This examination was made in view of the fact, as frequently recorded by English observers, that a painful tongue is one of the manifestations of gout.

A microscopic examination of the blood revealed a very slight secondary anemia, but nothing to suggest the pernicious type. She had some fever, with diurnal variations up to 100° F.

Since this examination six months ago, however, a great change has taken place. You will observe that in her present condition she is still well nourished, but note the marked anemia, the slightly yellowish tint of her skin, and the edema of her ankles. She has dyspnea on slight exertion, and she will tell you that even the slightest effort leaves her exhausted.

The blood examination shows for the first time a really grave anemia. The red cells number 2,500,000, and there is a marked anisocytosis and poikilocytosis; there are many large red cells (macrocytes), and the white cells, numbering 6000 to the cubic millimeter show a preponderance of lymphocytes. The hemoglobin is 50 per cent. No nucleated red cells have been found. The platelets have been counted, and are markedly diminished, being about 150,000 instead of about 300,000, as normally occurs.

In the urine there is slight albuminuria.

The nervous system itself appears unchanged, but the ophthalmoscope reveals hemorrhages in the retina.

The condition of the patient as you see her today is one of a very rapidly progressing anemia in spite of a very thorough course of treatment with arsenical medication and repeated transfusions.

The diagnosis has been made of *pernicious anemia*, based upon the course of the disease, the patient's general nutrition, the hemolytic type of anemia, and its special features, which we will discuss later. The prognosis is extremely grave. Note

especially the history of soreness of the tongue, lasting one and a half years without coexisting anemia.

Case II.—Before discussing this case, however, I would like to show you a second case, a man thirty-eight years of age, whose history of illness dates back to fourteen months ago, when he was thought by his doctor to be suffering from posterolateral sclerosis, the diagnosis having been based upon the paresthesia of his hands and feet, the ataxic gait, and the general weakness.

At this time there was no pallor or evidence of pernicious anemia, and the doctor's investigation at the time revealed no cause for the existing trouble.

For one year after the onset of his illness there was a gradual progressive development of the disease in his organic nervous system, but at that time the blood condition revealed nothing more than a very marked type of secondary anemia. But look at the patient today, four months after the last examination of his blood was made! You can readily see from the blanched appearance and the yellowish tint of his skin that now, at all events, a very serious anemia does exist. As a matter of fact, the red cell count is less than 2,000,000. There is an extreme grade of poikilocytosis, and the variations in the size of the cells is from an extremely small microcyte to that of numerous large macrocytes. Nucleated red cells, of all sizes, are present in abundance. The hemoglobin is between 25 and 30 per cent. The leukocytes are 5000 in number to the cubic millimeter, lymphocytes preponderating. The nervous system, however, does not show any longer evidence of posterolateral sclerosis. On the contrary, there is, in addition to the subjective symptoms in his hands, a very definite transverse myelitis. There is almost complete loss of sensation below the level of the twelfth dorsal vertebra, and complete paralysis of both the legs. His organic reflexes are seriously disturbed, and he has involuntary defecation. Moreover, it has been found necessary to catheterize him several times daily in order to avoid overdistention of his bladder.

Careful *x*-ray pictures of the spinal column have revealed no sign of vertebral involvement and no evidence of malignant growth.

Here, again, the diagnosis of pernicious anemia has been made, and the interesting feature is the long history of nervous manifestations prior to the onset of the anemia.

A careful examination has revealed no appreciable cause for his anemia; there are no foci of infection; there is no evidence of malignant disease, nor is there any cause for a grave secondary anemia. He has, indeed, all the classic features of a cryptogenic hemolytic anemia.

In this connection I would like to mention 2 other cases which came under my observation within recent years:

Case III.—A male, fifty-eight years of age, who for some months suffered from slight general weakness and loss of weight, and gradually developed a sallow complexion. At the end of six months there was no alteration in his blood examination. Only after about ten months, however, did any evidence of pernicious anemia appear. From that time on to his death, some three months later, two features were noteworthy:

1. The rapid development of a grave hemolytic anemia.
2. A very marked atrophy of all the muscles of his extremities, such as one sees in a chronic poliomyelitis.

The postmortem revealed a very definite pernicious anemia.

Case IV.—The other case was not a pernicious anemia, though the blood-picture resembled it. A young man, aged twenty-seven, was admitted to the hospital with a very grave anemia characteristic in all respects, so far as the blood-picture goes, of pernicious anemia. An examination revealed, however, a most foul condition of his teeth, with many abscesses at the roots. No other evidence of disease was present, and the removal of his teeth and the clearing out of the abscesses was followed by a very rapid improvement. One year later he returned on a visit to the hospital, when he showed every evidence of complete health.

Discussion.—The group of cases just exhibited illustrates in rather an interesting way some of the varieties that may be encountered in the onset and course of this disease. They present symptom complexes. After all, one must remember that *pernicious anemia is not so much an essential disease entity* with one definite etiology, but rather it is an expression of a *widely disseminated poison manifesting itself at various points of attack in the body, and in different ways.*

Pernicious anemia is not really a primary anemia; it is always secondary to some agency that affects the bone-marrow chiefly, destroys its erythropoietic myeloid function, causing hemolysis and preventing adequate blood regeneration. There is an upset in the relation between blood demand and myeloid function. Normally the equation should read thus:

$$\frac{\text{Blood Demand}}{\text{Myeloid Function}} = 1.$$

A true primary anemia must be a primary idiopathic disturbance of the primary blood-forming function, and such a thing, of course, does not exist.

One must look, then, upon pernicious anemia as having some unknown but definite etiologic factor of the nature of an infection or a toxin which acts generally upon various parts of the body, affecting always the bone-marrow and peripheral blood-picture, sometimes attacking the spinal cord in varying degrees, usually affecting various organs and tissues of the body, and thus providing a series of clinicopathologic features which may be arranged in groups as follows:

1. **Gastro-intestinal.**—Glossitis, stomatitis, achylia gastrica, and diarrhea (absence of hydrochloric acid is a constant feature of disease). Whether the alimentary canal is a seat of origin of the poison or not is undetermined. The blood changes do not seem to run concurrently with the gastric symptoms.

2. **The Nervous System.**—Evidences of a spinal sclerosis (numbness and tingling of the hands and feet; pathologic reflexes, sometimes spasticity, more rarely a wider spread of the

disease to other parts of the cerebrospinal tract). Mental disease is uncommon. Nerve and cord changes not infrequently precede the anemia, as in 2 of our cases.

3. **Blood Changes.**—I have noted on the board the essential features that characterize the changes of the blood. It must be remembered, however, that we are not prepared to say that the blood-picture is ever pathognomonic of pernicious anemia. The chief feature is, however, a marked hemolysis.

A. Red cells less than 2,000,000.

1. Variation in size (macrocytes, microcytes—anisocytosis).

2. Variation in shape (many tailed cells and other forms—poikilocytosis).

3. Variation in staining qualities (polychromatophilia, vacuoles, etc.).

4. Reticulation of the red cells suggesting immaturity and active bone-marrow.

5. Nucleated red cells (megaloblasts especially, not necessarily pathognomonic).

6. Occasional blastic crises (not pathognomonic).

7. Cabot's bodies and Howell Jolly bodies (disturbed hematopoiesis).

B. Blood-platelets diminished (diminished activity of bone-marrow); fragility increased.

C. White blood-cells diminished, with relative lymphocytosis (when slight leukocytosis occurs it implies some concurrent infection).

D. Hemoglobin relatively increased (high color index).

4. **Visceral Changes.**—Resulting from hemolysis:

(*a*) In the heart—Fatty degeneration; enlargement; murmurs.

(*b*) In the liver—Siderosis; enlargement; tenderness.

(*c*) In the spleen—Enlargement.

(*d*) In the kidneys—Siderosis; albuminuria.

5. **General Signs.**—Fever, weakness, dyspnea on exertion. Relatively slight loss of weight.

6. Add to these the course of the disease, with its insidious onset, progress with remissions, and the ultimately grave prog-

nosis, and you have a fairly characteristic picture of this strange cryptogenic anemia, idiopathic, pernicious, or addisonian, as you choose to call it.

7. I referred to the anemia as "hemolytic." *What are the signs of hemolysis?*

(1) In the blood—Plasma is yellow, with a green fluorescence, and the cell elements are distorted, as above described.

(2) In the skin and sclera—A yellowish tint, and the conjunctival fat of an unusually bright yellow color, as, indeed, is the body fat elsewhere.

(3) Urobilin in excess in urine, stools, and duodenal contents.

(4) The liver enlarged because of fatty degeneration and of pigment deposits (siderosis).

(5) The spleen enlarged from chronic hyperplasia and siderosis.

(6) The kidneys—Pigmentary deposits and albuminuria.

(7) Fever.

Differential Diagnosis.—There is little doubt that the blood-picture is not pathognomonic, and is seen in at least a few other maladies. Many other aspects must be observed to make the diagnosis, and many diseases must be excluded in considering the existence of the malady.

I will show you the list of a few in which, from one cause or another, pernicious anemia may be suspected:

Chronic hemolytic jaundice,
Chlorosis—aplastic anemia,
Addison's disease,
Recurrent hemorrhages,
Tapeworm,
Myxedema,
Lues,
Spinal cord disease,
Gastric cancer,
Cardiovascular disease,
Tuberculosis,
Sepsis.

The dictum still holds today that the blood-picture is not pathognomonic *per se;* that where no etiologic factor is found, however, and the course of the disease is characterized as above; where, in addition, the type of anemia is "hemolytic" the diagnosis of idiopathic or pernicious anemia may be made.

Treatment.—I will only refer here to the treatment in so far as transfusion and splenectomy are concerned. You are already well aware of the various forms in which arsenic has been used with a moderate degree of success, and I would like to dwell for a moment upon the importance of hydrochloric acid to counteract the effects of the achylia. There is no doubt that hydrochloric acid, given in sufficiently large doses for a sufficiently long time, will do much to help the alimentary canal, to improve the digestive power of the gastric mucosa, and to obviate the tendency to diarrhea.

You have also seen in former cases how the administration of salvarsan in one form or another has given benefit, more or less temporarily, and how the tendency has been to supersede this by means of transfusion.

Transfusion in Pernicious Anemia.—Transfusions in grave anemia are nothing new, and evidence goes to show that the practice is centuries old.

The question may be asked, What are the essential objects to be sought in transfusion?

The answer is clear:

1. The transfused cells remain alive as such in the circulation of the recipient. They live physiologically, which seems to account for the rapid improvement in many cases of marked anoxemia. It is thus a form of cytotherapy.

2. It revives deficient function.

3. It replaces dead cells that are killed physiologically, *e. g.*, in CO poisoning.

4. The injection of plasma helps in a biologic way in view of the biologic importance of the plasma.

5. From the point of view of physics the transfusion is of value through the isotonicity, density, and viscosity of the transfused blood, which thus replaces the defect in an ideal way,

and helps to revive tension, to maintain vascular equilibrium, and cellular activity.

6. The anticoagulating effect of citrate solution on the plasma is soon lost, and the power of coagulation is rapidly restored.

7. There are certain antitoxic and antibacterial properties of transfused blood worthy of consideration. And lastly,

8. There would seem to be some kind of organotherapeutic effect by which bone-marrow is stimulated, thus adding to the erythropoietic activity.

When shall we elect to transfuse in pernicious anemia?

(In the first place, one should never transfuse, except in extreme emergency, unless the compatibility of the blood between the donor and the recipient is tested.)

1. Only after focal infections have been removed.

2. Where remissions are frequent.

3. At the time improvement seems to be on the way.

As a rule, it is less beneficial to transfuse when the patient with pernicious anemia is very ill, or at the time of exacerbation, or during blood crises.

Dose and Repetition of the Transfusion.—Two opinions exist:

1. To give massive doses (1000 c.c. or more) at long intervals—two to four weeks.

2. To give smaller doses (200 to 400 c.c.) to counteract any anoxemia at first, and then repeat at shorter intervals (five to ten days), and as improvement follows, to lessen these amounts and lengthen the intervals between the days.

There is no doubt that in many cases a very few cubic centimeters will suffice, once the red cell count is stabilized.

Do not expect too brilliant a result from the first transfusion, because a marked improvement frequently comes only after several treatments have been administered.

The Results of Transfusion.—1. Improvement in the symptoms—noises in the ear, the vertigo, the nervous symptoms, and the general condition are all improved.

2. There is a gradual rise in the blood-count.

3. The remissions are longer, often lasting for several years.

Splenectomy in Pernicious Anemia.—The importance of the spleen in diseases of the blood must be kept in mind, as also its relation to the marrow, lymph-nodes, reticulo-endothelial apparatus, and the liver.

I will summarize some of the important features to be remembered:

1. The spleen is enlarged in various blood diseases.

2. It plays an important part in the destruction of red blood-cells in pernicious anemia.

3. Hemolysis is definitely associated with faulty splenic function. On the other hand, it is extremely doubtful if the defective function of the spleen is the *primary* cause of the hemolysis.

4. In hemolytic anemias the spleen contains many disintegrated red cells. Whether the spleen brings about this destruction or is merely a recipient of destroyed cells, which pass later on to the liver, is not determined.

5. The removal of the spleen in hemolytic anemias is often followed by marked improvement. The hemolysis diminishes and the urobilin excretion is diminished.

6. The effect of splenectomy in pernicious anemia sometimes results in marked benefit, but the effect is by no means so uniform and striking as in some other forms of hemolytic anemia (*e. g.*, hemolytic jaundice).

The immediate results are:

1. Improvement in the symptoms and general condition.

2. Improvement in the red cell count.

3. Diminished urobilin in the duodenal content, in the feces, and in the urine.

4. Increase in the number of nucleated red cells in the peripheral blood.

Complete cures are apparently rare, but life is often prolonged.

When should we attempt splenectomy?

1. Only after focal infections have been removed.

2. When transfusions have already been tried and found beneficial.

3. Preferably during a remission and when the blood is in relatively good condition.

4. Probably the earlier the splenectomy, the better.

When should we not attempt splenectomy in pernicious anemia?

1. In the aplastic type of anemia.
2. Where the spinal cord changes are marked.
3. Where transfusions have failed to give benefit.

CLINIC OF DR. G. GORDON CAMPBELL

MONTREAL GENERAL HOSPITAL

ACNE VULGARIS

ACNE vulgaris is a disease of the sebaceous glands, whose structure and association with the hair-follicles have already been brought to your notice. Clinically, it consists of a sprinkling of papules and pustules, often limited to the face, at other times extending to the back and front of the upper part of the thorax, and sometimes confined to these localities. It is a disease of adolescence, making its appearance at puberty, and tends to subside spontaneously about ten years later. It is invariably associated with the occurrence of comedones—blackheads in common parlance—and the initial step in the disease is the production of the comedones. Given the presence of comedones in the skin, sooner or later one or more becomes infected by pyogenic organisms, and a pustule is produced having the comedo as its center and showing on the skin as a small, brilliantly red papule, with the plugged orifice of the follicle at its apex. Once pus infection has occurred it spreads rapidly, producing lesions similar to that described, or another type whose method of formation is as follows: In many comedones the opening of the follicle is extremely minute and may be obliterated entirely by the pyogenic inflammation following infection, in which event the pus formed does not point at the opening of the follicle, but produces a small abscess within or below the skin, and gives rise to a large flattened-out papule very slightly raised, and covering an indurated area often reaching the size of a 5-cent piece. Invariably, however, if such a papule be pierced and the pus evacuated, the comedo is found in the contents of the pustule, softened and partly disintegrated, but

easily recognized. In this form of acne lesion, when the pus from the abscess is not evacuated, the inflammation in the course of a few weeks dies down, the fluid portion is absorbed, and the remainder becomes inspissated; but the destruction of the tissues surrounding the abscess leads to a falling in of the overlying skin in the form of a small pit or depression in the surface closely resembling a smallpox pit, but differing

Fig. 292.—Acne.

from it in that its floor is not cicatricial tissue. Where most of the lesions are of this type the disease is often called acne varioliformis.

Having seen that every acne lesion contains a comedo, it follows that if there are no comedones there can be no acne; and, furthermore, that if we can prevent the formation of comedones and remove those already present, we can cure acne.

This leads us to inquire into the origin of comedones, and here we are on debatable ground. The comedo consists of retained and dried sebum filling the duct of the sebaceous gland, which is the hair-follicle. The sebum may be retained either because it is too thick to flow out of the orifice of the follicle or because there is an obstruction to its escape. Most of the evidence points toward an obstruction at the orifice, but not necessarily due to a narrowing of this orifice. Many comedones are bottle necked in shape, the small end being at the orifice of the follicle, and

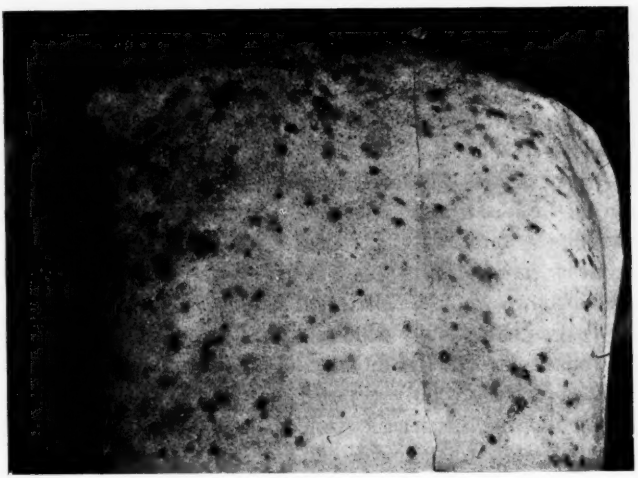

Fig. 293.—Acne.

you can readily understand that only obstruction at the orifice could give rise to such a phenomenon, the sebum at the opening is arrested, hardens, and the fluid sebum dilates the duct below, and it, in turn, becomes dry and firm. What causes the block at the orifice, is it an inflammatory reaction or a mechanical one? Clinically there is no evidence of inflammatory reaction, the skin surrounding the comedo being of normal color and consistence. Bacteriologically, when the contents of an acne pustule are examined, the organism most commonly found is the Staphylococcus albus, next the Staphylococcus aureus,

and lastly the Acne bacillus. While the staphylococci are plainly the cause of the inflammatory disturbance, may not the acne bacillus be responsible for the comedo either by narrowing the size of the orifice or by altering the consistency of the sebum? Such a view is held by some dermatologists. When we find, as we do, many comedones of cylindric shape as large as the lead of an ordinary lead pencil, this explanation becomes untenable. My own opinion is that two factors enter into the production of comedones, one being a lessened fluidity of the sebum, and the other, and the most important one, blocking of the orifice by dirt. The evidence in favor of this is quite strong. Many people wash the face at long intervals only, once a day at most, and there is a popular idea that soap should not be used on the face. Again, in women who wear their hair over the forehead and on pads in front of the ears, comedones form in much greater numbers in these situations. Still stronger evidence is seen in the fact that in individuals having deep transverse furrows in the forehead these are often found marked out with a row of comedones at the bottom of the furrow, and many other instances could be cited.

We are unable to say definitely what is the cause of the thickened consistence of the sebum. We do know that comedones rarely occur before puberty, and that girls under twelve years of age showing numerous comedones are also precocious in the matter of sexual maturity, as evidenced by the establishment of menstruation. It seems reasonable to infer that with the increased activity of the glandular system, known to occur at puberty, there may be sufficient alteration in function to cause a lessened fluidity of the sebum.

In considering the treatment of acne there are two main indications: First, we must get rid of the comedones already present and endeavor to prevent the development of new ones; and second, we must deal with the pus infection. Failure to permanently cure the disease almost invariably results from neglect of the first indication. Comedones cannot be removed by applications made to the surface of the skin with any greater success than one would have in trying to extract a cork from a

bottle by treating the surface of the glass. Pressure applied around the orifice of the duct, however, will cause the plug to pop out of the follicle, and an instrument known as a comedo extractor has been devised for this purpose. Unfortunately, most instrument makers, striving to combine two instruments in one, make a small cup-shaped curet in the bowl of which a small perforation is placed to act as a comedo extractor. The rounded bulb of the curet defeats the purpose for which it was devised by lessening the pressure that can be applied. The old-fashioned watchkey is most effectual, but apt to be painful, and tends to cut the skin because of the small amount of metal surrounding the opening in the barrel of the key. Stimulation of the sebaceous glands with resulting greater fluidity in the sebum can be brought about if one increases the flow of blood to the skin, and this is accomplished by bathing in water as hot as can be borne or steaming over the vapor from very hot water. At the same time a thorough shampooing of the skin with a non-irritant toilet soap will remove the overflow of sebum which collects in the furrows, and keep the orifices of the ducts open. This should be carried out at least once a day; and we find, after a week of such bathing, that the lower parts of the comedones, formerly hard, are softened and more easily expressed.

In treating the pus infection it is imperative to evacuate all pustules as soon after they are formed as possible, to prevent further infection. Those pointing at the surface are readily evacuated by pressure with the comedo extractor, when both the pus and the comedo are forced out, and slight bleeding occurs. It is not sufficient to simply open the pustule and allow the pus to escape, if the whole of the comedo is not removed the small abscess will re-form. In opening those pustules which do not point superficially it is generally possible with a strong lens to see the spot in the skin corresponding to the opening of the infected follicle, and the needle should be inserted here, when the contents plus the comedo can be evacuated by pressure with the extractor. Opening the abscess at random often results in some difficulty in delivering the comedo, and the

pus collects again. I have never found it necessary to curet the cavity of the small abscess, a procedure recommended by some dermatologists.

The needle found most satisfactory for the purpose is a straight Hagedorn, which can have the tapering portion of the point sharpened on one side to a knife edge. Alcohol is found to be the most reliable antiseptic, and is used freely during the evacuation of the pustules, and the patient directed to swab the part with it after the accidental breaking of a pustule and to wipe the face with it after the hot wash.

In cases of moderate severity the duration of the treatment depends upon the time it takes to get rid of the comedones, and that, of course, upon their number and how often one works at expressing them, for the physician must do most of that part of the treatment himself. In cases showing a large number of pustules, and especially when these are of the blind variety, the duration of the treatment can be very considerably shortened by measures directed toward prevention of pus formation or destruction of pus organisms *in situ*. To reduce pus formation we use bacterial vaccines, either stock preparations or preferably autogenous, made from the patient's own pus. After considerable experience with mixed vaccines containing the staphylococci and the acne bacillus we have abandoned them for a vaccine made from the organism found in the pus, which is almost invariably Staphylococcus albus. We use it in comparatively large doses, giving 500,000,000 in the first dose and following with 1000 and 1500, with a week's interval between the injections. It is unwise to keep on administering the vaccine after the third dose, though some months later, should anything interfere with the course of the treatment, it can be used again. Unfortunately in hospital practice many people get so much temporary benefit from the vaccine that they cease attendance before the comedones are all removed, reinfection occurs, and one has to do the work over again. It is imperative to evacuate the large pustules regularly while administering the vaccine. Failure to do so results in the small abscesses becoming encapsulated by an area of indurated inflammatory tissue which is

most unsightly, forming hard, dark red lumps in the skin. These collections of pus ultimately, perhaps after several months, break down and discharge their contents.

The methods used for destruction of the pus organisms in the skin consist of the Roentgen and ultraviolet rays. Both have their advocates, in fact, this method of treatment is the most popular one at the present time. They have several disadvantages: first, that they have to be used in dosage large enough to produce a severe dermatitis in order to bring about the desired result, and the patient is consequently incapacitated for a time; second, that their use does not do away with the necessity of removing the comedones and evacuating the pustules. It too often happens that the physician who has at his command treatment by either of these two agents is disinclined to carry out the tedious process of removing comedones and opening pustules, and the reappearance of the disease is only a matter of time

You will notice that I have not alluded to the necessity for careful dieting and the treatment of gastric disorders on which so much stress is laid in the text-books. That is because I believe gastric disturbances play no part in the production of acne. In twenty years' experience as a physician to this hospital I was unable to find any evidence pointing to a connection between the two conditions. In this clinic we rarely now inquire into that part of acne patients' history, we never make any effort to regulate their diet, and we have great success in our treatment of the disease. Where, formerly, the appearance in our clinic of a patient demanding treatment for acne was more or less dreaded from our helplessness as regards a cure, we now enter confidently on the method of treatment outlined to you, and the number of cases applying for aid has increased out of all proportion to the general increase of the clinic, which fact is perhaps the best evidence of the results obtained.

CLINIC OF DR. A. T. HENDERSON

ROYAL VICTORIA HOSPITAL

ON THE SUCCESSFUL TREATMENT OF ASTHMA AND RELATED CONDITIONS

IN this clinic I shall attempt to present to you a series of cases which illustrate some of the phases of protein sensitization as observed by us in the study of asthma and related conditions in the asthma clinic of the Royal Victoria Hospital.

All the cases which I shall mention have been tested by the cutaneous or scratch method, to a routine list of proteins, comprising the following: lactalbumin, casein, egg-white, egg-yolk, potato, bean, pea, barley, oat, rice, wheat globulin, beef, chicken, pork, lamb, goose feather, chicken feather, horse dander, and timothy and ragweed pollen. Where indicated, cat hair, dog hair, rabbit hair, sheep wool, etc., have also been used for testing, and frequently additional food proteins.

Furthermore, practically all of these cases have been observed over a fairly long period of time, and we feel that we can lay some claim to their being proved cases.

FOOD ASTHMA

First we shall consider food asthma:

Case I.—W. J. C. is a boy who is now thirteen years old. He first came to our attention October 7, 1921, when he was admitted to the Children's Ward, service of Dr. Fry, complaining of bronchial asthma, frequent vomiting, and attacks of urticaria.

The history as obtained from his mother was that he had had attacks of bronchitis and asthma since infancy. He was nursed during the first year and he had "bronchitis" all the time. Asthma set in after this and has continued until the time of admission. Attacks recur frequently, are rather severe, and seem

to come more often in the winter months; but in August of this year (1921) he had a bad attack and was in bed a full week.

The attacks start with wheezing and tightness in the chest, he can't breathe, and "he does not like to have his stomach touched." There is no cough at first, but later on a paroxysmal cough develops, as if he wanted to get something up and couldn't dislodge it. Finally, he raises thick white phlegm "in pieces," and then feels easier. Vomiting may occur. The attack lasts off and on for two or three weeks, and leaves him quite exhausted. On this account he has had to miss a lot of school. Between attacks he is, as a rule, quite free from dyspnea or cough; although his mother thinks that going out on a cold winter's day may have had something to do with precipitating some attacks, while special exertion has seemed to make him short of breath.

For years there has been a frequent history of vomiting which has never been accounted for. Almost regularly he has had vomiting in the morning after breakfast, which generally consisted of half a cup of tea with milk and sugar and bread and butter. Often vomiting is preceded by a pain which starts in the pit of his stomach and mounts up to his head. "Hives" have for years been a great plague to him, occurring frequently, "nearly driving him mad" sometimes.

The important thing in the personal history is that at the age of three months he commenced to have eczema, which lasted for eighteen months.

On admission there were scattered dry râles throughout the chest, with some generalized hyperresonance and prolongation of expiration. Temperature, pulse, and respiration were not disturbed. Nose and throat examination by the specialist was negative, as was the *x*-ray of the chest. Von Pirquet tuberculin test gave no reaction. The blood-count showed Hb. 75 per cent., R. B. C. 4,300,000, W. B. C. 13,000, the differential count being essentially normal. Eosinophils were 4 per cent.

His protein tests showed as follows:

Wheat globulin, +(+)
Barley, +(+)
Rice and egg gave + reactions

In this, and all other cases, a one plus reaction consists of an urticarial wheal ½ cm. in diameter, with a surrounding zone of erythema. Two plus would be 1 cm. in diameter, etc. In addition to the other routine proteins, he was negative to wheat glutenin, wheat gliadin, and lobster. It is to be noted that oat failed to give a reaction, but he reacts mainly to the grains. The test to wheat globulin has been repeated on several occasions since, and is constantly positive.

On October 9, 1921, two days after admission to the ward, no change or restriction having been made in the diet, the note appears: "Last night slept well until 4 A. M. Then he woke up complaining of severe headache and nausea, and he vomited a large amount of clear fluid. No pain or diarrhea." On inquiry, it was found that for supper the night before he had baked apple, bread and butter, and milk only, cream of wheat having been refused.

He was discharged from the ward on October 15th on a diet to exclude wheat, barley, and eggs. On October 24th he returned to the outdoor department, having had dyspnea and wheezing, with cough, and vomiting for an entire week. He had, however, not been observing instructions as to diet.

On October 31st he again visited the clinic, having had a bad night of asthma a few days previously. Dry râles were present throughout the chest; and as he was one of our first cases, he was again taken into the ward for further tests and observation. The only additional protein found positive was corn. During a two weeks' stay on strictly observed diet he was entirely well. As there was no sputum, a vaccine was prepared from a throat swab, but this was never employed.

He was discharged on November 14, 1921. Since that time, but for an occasional wheeze, he has had no recurrence of asthma nor any return of vomiting, except in October, 1922, when he had both asthma and vomiting for two days after he had been eating bread for two weeks. This was a good object lesson to the mother and to the boy; and they decided to get along without wheat, except crisp toast, shredded wheat biscuit, and corn flakes, which can generally be taken without trouble. As oat

had been found negative, he was advised to try oatmeal muffins in lieu of bread. It was no hardship to do without barley and corn, and he didn't like eggs. Rice was also to be excluded for the time being.

From time to time, however, this boy, although he has had no asthma, urticaria, or vomiting, has appeared at the outdoor department with frequent colds, not infrequently on the chest, with some cough, and the presence of dry râles at times. On investigation, it was found that his bedroom admitted no fresh air in the winter months when the double windows are on. This was remedied, and his mother was induced to try cool morning sponges to his throat and chest. Since then he seems to have been much freer from infectious colds, he is a much better color, and at times has eaten sparingly of stale bread. This, however, is for the present not encouraged.

Discussion.—We can learn a good deal from this case. In the first place, his asthma dates from infancy, which would make us think that he belongs to the protein-positive group, even before we have done our tests. Walker found that four-fifths of his cases of asthma that began in infancy were protein positive. Furthermore, we could hazard the guess that it is due to a food protein, as it is well known that the coexistence of eczema and asthma in infancy points in this direction; and this boy had both; and, in addition, his frequent attacks of urticuria indicate the same thing.

Duke and others have shown that a manifestation of food allergy may be abdominal pain, with nausea and vomiting, due to a specific sensitization of the gastro-intestinal mucosa. Where the attacks occur infrequently it is generally due to some uncommon article of food. If the attacks are frequent the patient may have chronic indigestion, and it is probably some food which is commonly eaten.

Now Walker found in his analysis of 600 cases of asthma that one-half of these were sensitive to some protein, and of these, that 74 cases were sensitive to food. Of these 74 cases, 36 were due to cereals, 25 being to wheat alone, 3 to corn alone, and 2 to rice alone, 6 reacting to all three of these grains. Of

the 38 cases sensitive to other foods, 16 were due to egg, 5 to milk, 9 to fish, and 8 to potato.

It is, therefore, not surprising to find that this boy is sensitive to wheat, which in the form of bread is eaten every day; and explains his recurrent vomiting and frequent attacks of asthma.

Case II.—H. J. F., male, aged thirty-seven years. Admitted to a medical ward November 16, 1923.

Complaints.—Attacks of difficult breathing, with cough; and pain in the abdomen on exertion.

History of Present Illness.—The condition began two years ago and followed a "severe" cold. The first attack was characterized by great dyspnea and weakness, and lasted about two weeks, he says. There was no cough, and he managed to keep at work, as foreman in a raw sugar warehouse, a very drafty structure.

From November, 1921 until March, 1922 he was subject to minor attacks of asthma lasting two or three days, with more or less acute shortness of breath, and accompanied by cough, which disappeared on the cessation of the attack. He also had frequent head colds, lasting a few days, but he continued at work all winter.

In March, 1922 he had a second severe attack, lasting over a week. After this, until November, 1922, his breathing was practically never free. He kept at work, but at night seemed exhausted and his sleep was poor because of dyspnea. Cough was then not persistent, but was more or less spasmodic, lasting a few days at a time. In November, 1922 he was forced to take to bed for two weeks because of an exacerbation of symptoms. He returned to work at the end of that time, and remained at work until October, 1923, except for two weeks in April.

In spite of medical treatment by his own doctor he has continued to have periodic attacks throughout the year. Cough and expectoration have been present continously during the past two months, especially worse at night, and are dry and hacking

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at the height of the attack, and looser at the end of attacks. He has been unable to work for over a month. His breathing is now noisy and wheezing.

Personal History.—Negative, except for history of his being subject to periodic attacks of bronchitis as a youth.

Family History.—There is a strong family history of this condition, an uncle on his father's side, one sister, and his brother's child all having had asthma, while his maternal grandmother was said to have had asthma all her life.

On admission he had a hacking cough, his breathing was wheezy, and sputum copious. Dyspnea appears to be inspiratory rather than expiratory. Chest somewhat emphysematous, with slight generalized hyperresonance. Expiration is greatly prolonged, and numerous rhonchi are heard during both phases. Nothing else of moment was found on physical examination. The *x*-ray of the chest showed intensification of the pulmonary roots and bronchial tree to apices and bases. No fever during stay in the ward.

The differential blood-count was interesting in showing 19 per cent. eosinophils. The sputum was negative for tubercle bacilli, showed many pus-cells, contained no spirals or crystals. *Streptococcus hemolyticus* and *Staphylococcus albus* were grown in the culture, and a vaccine was prepared.

Protein tests showed marked reactions to the grains and other proteins as follows:

Wheat globulin,	+++++	Peas,	+++++
gliadin,	++	Beans,	++(+)
glutenin,	+		
Oat,	++(+)	Celery,	++
Barley,	++	Tomato,	++
Rice,	+(+)		
Rye,	+(+)	Goose and chicken feather,	+
Corn,	++	Timothy and ragweed	(+)

All other routine tests were negative, also codfish and salmon.

He had no attacks while in the ward on a diet based on the above, and his chest became free from râles. Two days after discharge (November 16th), in spite of strict dieting, he began

again with wheezing, cough and asthma, and had abundant sputum.

On December 3, 1923 we began to give him weekly inoculations with his autogenous vaccine, commencing with $1\frac{1}{2}$ m. (1 c.c. = 500,000,000) and increasing by $\frac{1}{2}$ m., and then by 1 m. each week. He has had no asthma since. He has kept steadily at his work in the sugar warehouse. There has been slight cough and slight wheezing on exertion only. He has gained 13 pounds in less than ten weeks.

March 3d: Retested with pollens. Timothy and ragweed + (just), goldenrod negative, sunflower + (+).

Discussion.—In this case the history points so clarly to bacterial infection that in spite of very marked and clear-cut reactions to the grains and other foods, we felt at the outset that vaccine therapy would be required in addition to omitting the foods found positive. To begin with, the condition commenced two years ago following, he says, a severe cold; from March until November, 1922 his breathing was practically never free, and exertion made it worse. Finally, for two months prior to admission, cough and expectoration were continuously present.

We were, therefore, not surprised when, in spite of carefully following dietary restrictions imposed, he returned to the outdoor department on November 15, 1923, having had a return of his symptoms. It is remarkable that from the commencement of the use of the autogenous vaccine, in spite of his working in an unfavorable environment, he has continued to make steady progress, and has been free from any but the mildest symptoms.

Since writing the above the patient has again been seen at the outdoor department on March 10, 1924. On the night of March 7th his breathing began to be difficult; this developed into asthma during the afternoon of the 8th, and was worse on the 9th, when he stayed at home all day, as it was Sunday, but continued to have asthma. On March 10th, though easier, he is still dyspneic. On inquiry, we found that he had had pea soup on the night of March 6th, strained, it is true; and also that he had eaten fresh bread the day before, both of these foods being

the very worst things he could take, judging from the extent of the skin reactions.

We therefore feel that here we have a case of food asthma, complicated by bacterial infection of the bronchial tract, and that infection was probably the precipitating factor.

The next two cases are sensitive to foods, but in them the asthma is due not only to the ingestion of the offending protein but also, and possibly principally, to its inhalation as dust particles.

Case III.—Mrs. E. B., aged thirty-nine, was admitted to the gynecologic ward in April, 1922 for amputation of the cervix, etc. Following her operation she had a very severe attack of asthma which lasted a full three days. Her history was that she first had an attack of asthma at the age of fourteen years in London, England. Up to 1905, when she was twenty-two years old, she had had three attacks. From 1905 until 1910 she was free. In 1910 she came to Canada, and she has had asthma ever since, with the exception of 1914 to 1919, when she returned to England. During this time she had only one attack, and then had her tonsils removed. On her return to Canada in 1919 asthma again became marked.

Her asthma is in the winter months, never in the summer, and attacks occur about every two weeks, and last generally about three days. This winter attacks are not so bad as formerly. The attack starts with tightness in the chest, is characterized by expiratory dyspnea, and there is cough only at the end, when she raises phlegm like "clotted flesh." There is no sneezing.

The *personal* and *family histories* are negative.

When first seen the chest was full of asthmatic râles, and expiration was much prolonged. She was just getting over the attack, which she says came on after eating beans for dinner; she has had her suspicion of peas. The protein tests showed the following results: Barley, + (+); oat, ++; wheat globulin, + (+); beef, +; horse dander, +; and bean, + only (doubtful), other tests being negative.

On discharge she was advised to exclude from her diet oat, barley, bean, and wheat, crisp toast being allowed, and to

return to the outdoor department. She replied that she never ate oatmeal, but that she made it every morning for the children, and had observed that whenever she opened the tin she would be caused some discomfort in breathing. The fact that the children got oatmeal only in the winter months, and never in the summer, accounts here, we think, for the seasonal periodicity of her asthma. She returned to the outdoor department May 22, 1922, having had no asthma except one slight attack the week before, after eating corn. Further tests were done with rye, corn, and wheat, all of which gave + reactions, and oat ++ (1 cm. wheal by 3 cm. erythema) as before.

She returned at the end of June, having been quite well. She had relegated the making of the porridge to her daughter; and volunteered the information that in cooking she had noticed that wheat flour made her sneeze and her eyes itchy. Since this time, whenever she makes pastry, etc., she always covers her nose and mouth with a cloth, and so escapes trouble.

On March 7, 1924 she returned to the outdoor department. She has been completely free from symptoms except when she made oatmeal porridge once this winter, and fifteen minutes later became very uncomfortable, and had dyspnea for two to three hours until she burned some powders.

Case IV.—J. F., male, aged thirty-eight years, first seen December 9, 1922.

Complaints.—Subject to chronic cough for years; and for the last month he has had attacks of difficult breathing.

Personal History.—He has been a cake baker for twenty-six years, and now has the position of a superintendent. For the last year he had chronic eczema on the right hand. Otherwise negative.

Family History.—Negative.

History of Present Illness.—For twelve or thirteen years he has been subject to very frequent coughs. One month ago he was treated by his doctor for "bronchitis," which lasted five days. On December 1st he had an attack of acute dyspnea, which his doctor said was asthma; it lasted only about ten min-

utes. Four days later, while at his work in the bakery, for many hours he had hard breathing, with harsh cough, and he raised some white frothy phlegm with difficulty. The next day he had full-blown asthma when seen by his doctor.

During the previous summer he had been troubled a great deal with sneezing. This, he said, began about May and continued until well on into November. Every day during the summer he had had sneezing, which only occurred during the mornings, and would always stop at noon.

The protein tests were done, and wheat globulin gave a + (+) reaction, all other tests being negative, including the pollens.

On inquiry it was found that his sneezing coincided with the time when the automatic sifters were running. It was considered that his symptoms were due not to the ingestion so much as to the inhalation of wheat. Accordingly, arrangements were made whereby he would not be exposed to the wheat dust; and since then he has been free from trouble.

His answer to our inquiry on March 8, 1924 was that since he had been protected for the most part from the wheat dust he had had no asthma, although he had made no attempt to limit the amount of bread, etc., in his diet.

Comment.—These cases are interesting as representing types of dust asthma.

In the case of Mrs. E. B. symptoms are probably due to ingestion as well as inhalation of oat and wheat proteins. In the case of J. F. the principal, if not the only, way by which symptoms are provoked is by inhalation of the wheat protein. This patient is also a good example of occupational asthma.

CASES OF EPIDERMAL ASTHMA

We now come to consider the subject of asthma due to epidermal proteins.

Case V.—G. M., a boy aged eleven years. Admitted to the ward on October 30, 1921.

History of Present Illness.—The asthma began at the age of four years and has continued ever since, except that during

recent summers he has been in the country and has then been quite well. The attacks last from eight to ten days, with short intervals averaging a fortnight, when he is perfectly well and free from cough. The attack is quite typical, the dyspnea being associated at first with dry cough, which later becomes productive. Sweating and fever has been noted at the height of attacks. The last attack of asthma began on September 2d, and his doctor says it was the worst he had ever seen in any patient. There was nothing in the history to suggest the cause except the fact that he had a pet cat at home of which he was very fond.

Personal and *family histories* negative.

While under observation there was slight fever to 100° F.; slight bronchitis on admission; *x*-ray of chest negative; Von Pirquet negative; urine negative. Protein tests showed horse dander, ++ (+); cat hair, ++; dog hair, +; wheat and oat, +; barley, (+); he was negative to all other routine proteins and, in addition, to buckwheat, cucumber, lobster, oyster, sheep wool, cattle hair, celery, and corn; also to the bacterial proteins.

He was tested with horse dander dilutions: 1 in 500, 1 in 1000, 1 in 10,000, and 1 in 100,000, both by cutaneous and intradermal tests; intradermally he reacted strongly to all dilutions. By the scratch method 1 in 1000 was positive, but 1 in 10,000 and 1 in 100,000 were negative.

We decided to eliminate the cat before considering treatment with horse dander; and this was done "with regrets." He was advised to exclude from the diet oat, wheat, and barley. On December 12, 1921 he appeared at the outdoor department having had no asthma or cough of any kind.

On September 1, 1923 (nearly two years later) he again came to the outdoor department, as he had had a bad attack of asthma for three days the week before after getting very wet. Six months previously he had also an attack, his mother stated, lasting for three days, but apart from these he had been perfectly well since discharge from the ward in November, 1921. At this time (September 17, 1923) he was retested with horse dander, cat hair, wheat globulin, wheat glutenin, oat, and rag-

weed, as this was the height of the ragweed season. Horse danger gave ++ (+); cat hair, ++; wheat globulin, + (+), and other proteins were entirely negative. He was again advised to restrict wheat to crisp toast, etc.

Note on March 7, 1924: Since September 23, 1923 he says that he has been wheezy occasionally, but has not been laid up at all. Once or twice he has had to stay away from school in the afternoons. For the last week or ten days the neighbor's cat has been boarding at his house. No dog. In regard to food, he has eaten no oatmeal or barley. As for wheat, he has been eating bread, generally a day old, and cream of wheat occasionally. He has had no vomiting. He was advised not to harbor the cat, and to eat no cream of wheat. Toast and stale bread allowed.

Comment.—Here we have a case of epidermal asthma, with "two plus" reactions to horse dander and cat hair, and a less strong reaction to wheat globulin. The primary factor appears to be epidermal; and in 1921, owing to the "imminence" of the cat, it seemed justifiable to deal with this first. The subsequent history seems to show that we were right in our surmise. If, however, there is a recurrence of asthma, which cannot be ascribed to "transient" cats, or to the too free ingestion of wheat, desensitization to horse dander may be called for. Such recurrence of symptoms as he has had can, we feel, be put down to the last two factors.

Our next case is one of epidermal, and probably, food asthma.

Case VI.—A. L., male, aged eleven years, was first seen December 4, 1923.

History of Present Illness.—The history dates from infancy. At the age of five or six months this boy used to get spells of heavy breathing, which were put down to tonsils. Not long after this he began to have recurring colds every couple of months, which came on like hay-fever, with sneezing, etc., and developed into bronchitis, so that at the age of a year he was having frequent attacks of bronchitis with wheezing. This condition continued until he was three and a half years old, when he was

operated on for tonsils. Three weeks later bronchitis again set in, associated with heavy breathing; and thereafter, about every two months, he had attacks of heavy breathing and wheezing on his chest.

In May, 1921, at eight and a half years, his mother moved to Winnipeg, hoping that the change of climate would bring about an improvement. He was well until the following August, when he had a severe attack of asthma, which began with sneezing and lasted ten days.

He was then free until December, 1921, when he had a mild attack. In February, 1922 he had an influenzal cold with slight asthma. He then remained completely well until August, 1923. At this time, following a motor drive, he caught a cold. The attack began with irritation of the eyes, sneezing, and discharge from the nose; then he developed cough and asthma which did not clear up for three weeks.

He and his mother returned to Montreal from the West October 28, 1923. He was quite well until one Saturday, early in November, he went to visit his grandparents in the Laurentians. He drove behind a horse from the station. That night he had chicken pie and hot buns for dinner. The next day he had asthma, which came on as "indigestion," with upset stomach, intermittent pain in the epigastrium, and vomiting. The attack lasted Sunday, Monday, and Tuesday, but he did not clear up entirely, and on November 26th, after another "dining out," he had indigestion and asthma, which lasted for three days. When seen by me on December 4th he still appeared short of breath. Generally, however, he has been free from cough and dyspnea between attacks.

Personal History.—No eczema or hives. Subject to attacks of indigestion. Measles and mumps; tonsils operated upon at three and a half and again at seven years.

Family History.—Negative.

Protein tests were done: Cat hair was + (+); goose feather, + (+); horse dander, +; rabbit hair, +; dog hair was (+), but itchy; wheat globulin, +. He was negative to other routine tests.

and to corn, tea, walnut, celery, tomato, salmon. Chicken feather was +, and timothy and ragweed were both negative.

On more careful inquiry, in the light of protein tests, it was found that before going to the West there were no pets in his home in Montreal, and that his asthma would always be worse on going to his grandmother's home in the Laurentians in the summer holidays, and also at Christmas, where there were always at least two cats; furthermore, that from the railway station there was a drive behind horses of two miles. In partial elucidation of the history when in Winnipeg we found that his severe attack of asthma in August, 1921 coincided with a visit to an aunt's in the country, where he was exposed to horses and dogs, and where hot buns were the order of the day. Now the attack which occurred in August, 1923 may be accounted for in this way. The cat in the house received but scant notice up to this time; but in August kittens arrived, and the family were then moved up to his room. It will be remembered that this attack began with irritation of the eyes and nose.

In the light of our tests, (1) he was advised to use a substitute pillow, (2) to avoid cats, dogs, and horses, (3) to restrict wheat for the present to toast, etc. On March 10, 1924 this boy again appeared. He has been perfectly well, with no symptoms whatever, except for a slight cold in his head. He is using a "Kapoc" pillow and cotton comforter. There are no dogs and cats in the house, and he is staying away from horses. He has eaten no wheat except toast until three weeks ago, since when he has been eating stale white bread and biscuits, with no trouble.

Comment.—This case is particularly interesting from the point of correlating the clinical history with the protein findings. It shows rather well the value of taking a careful and detailed history, which in a good percentage of cases will give a very definite clue to the line of attack. We were particularly fortunate, of course, in that his mother was an excellent witness.

A little later we mean to remove the embargo on wheat and watch if it makes any difference. Whether feathers have much to do with his asthma or not we cannot say at this juncture.

I now wish to present 2 treated cases of horse dander asthma, from an analysis of which we may learn some lessons.

Case VII.—J. P., male, first seen December 1, 1921 at the age of thirty years.

The history was that for the past five years he had been the subject of very frequent "colds" in the head, with much sneezing and copious nasal discharge, and on the chest, with heavy breathing, his dyspnea being intensified if he moved about. Very little cough. These colds are worse in the winter. He often has to take off his collar and lie down in an attack. In the summer he is generally quite clear unless he goes close to horses, when his eyes water and he has sneezing.

Personal History.—He is employed in a large grocery and provision shop, where the delivery wagons are practically all horse-drawn. The drivers are constantly in and out of the shop. No history of hay-fever; no eczema or urticaria. Dry pleurisy in May, 1921 following an operation for appendicitis. Typhoid fever at sixteen years.

Family History.—Paternal grandmother had asthma from the age of seven years.

The protein tests were done and showed the following results:

Epidermals.		*Foods.*	
Horse dander,	++++	Tomato,	++
Goose feather,	+++	Oat,	+
Dog hair,	+++	Pea,	+ (delayed)
Cat hair,	+(+)		
Sheep wool,	+		
Chicken feather,	+		

He was also tested to several of the bacterial proteins, but by some oversight we neglected to do the pollens.

Besides doing away with his feather pillow, it was decided to desensitize him to horse dander; 1/500 reacted almost as strongly as the whole protein, and 1/100,000 was +. We commenced treatment with 1/1,000,000 dilution, the Arlington Company's 1/100,000 being diluted 1/10 with $\frac{N}{100}$ NaOH.

In going over the diary of treatment I am struck with the frequency with which "colds" or "asthma" is mentioned, as occurring soon after the injection; and when inquired about, that often there was considerable local reaction set up. This was particularly so in the earlier half of his treatment, and I now feel convinced that his desensitization was too rapid. This probably accounts for the fact that the extent of his skin reaction to horse dander (at the close of his treatment) was not lessened appreciably. It cannot be stated too emphatically that if a treatment injection be followed by a considerable local reaction, or by general symptoms which are unexplainable from other causes, then that is a sign to increase more gradually, or not to increase at all, at the next injection.

The clinical result, however, appeared to be fairly good, as on May 4, 1922, when he received 6 minims of 1/500 dilution, the following note appears: "He has moved out into the country for the summer, and drives behind a horse from the station each day, for a short distance, with no symptoms, and his attacks of sneezing, which formerly were a source of constant annoyance, have disappeared. Furthermore, he states that the frequent nausea, which was present before his appendix was removed, and continued right up until December last, has now completely gone."

From May 4th until the end of June, when the treatment ended, no matter how slowly we increased the quantity, he continued to show excessive local reactions; and we put this down to the fact that in driving to his home every night he was also, of course, receiving horse dander through inhalation, and this was throwing us out on our calculations.

On March 7, 1924, nearly two years after his treatment ceased, he reports that he still gets his "head colds" a bit, but not severely. He has had no real one this winter or last winter either.

Last summer he drove home behind a horse half a mile every evening. He did not notice any effect whatever. He had no asthma or "hay-fever" all summer.

This winter, however, he has had asthma four or five times. These attacks have generally occurred on leaving the store

to go home, and going out into intense cold. He wondered if potatoes might not be the cause, as each time he had had one of these attacks he had eaten a good deal of potato for lunch. Potato was put on, and gave ++. Ragweed also gave ++. Timothy was negative. It would seem that these attacks may have been provoked by potato, and precipitated by sudden change of temperature. Accordingly, we advised him to eat potato in strict moderation and only well baked, as they can usually be tolerated in this way.

As for the ragweed, which gave a ++ reaction on March 7, 1924, since he had no symptoms last August and September, we are inclined to do nothing about this at the present time.

The second case of treated horse dander asthma was:

Case VIII.—S. B., male, aged thirteen years, first seen on October 14, 1922. His history was that for over seven years (from the age of six) he had suffered from asthma, particularly in the summer months. The first attack came on in the month of August while in the Laurentian Mountains, and it began with a "cold in the head." Since then attacks have recurred every summer. Last winter he had no trouble until April of this year (1922), when it began with sneezing, and soon developed into paroxysmal dyspnea, with wheezing and cough, but never any expectoration. His attacks continued on and off practically all summer, being perhaps worse in the months of July and August. They generally lasted about three days, and then would begin to ease off; but he would hardly be clear when the next attack would commence. Sneezing and nasal discharge were usually prominent symptoms at the beginning. During the last attack he vomited some fish, but not infrequently his stomach is upset during attacks.

Personal History.—He occasionally gets "hives," but no eczema. Adenoids removed five years ago. Last winter he gained weight from 59 to 69 pounds. No cats or dogs in the house.

Family History.—His mother had asthma as a child from the age of four years to about nine, and then none until she was twenty-one, when she had an attack for two or three weeks. Since then she has been free.

The protein tests gave the following results:

Epidermals.		*Foods.*	*Pollens.*	
Horse dander,	++(+)	None (?)	Red top,	+
Dog hair,	+	Vomiting fish	Daisy,	+(+)
Cat hair,	+	Hives (?)	Timothy,	(+)
			Corn pollen,	+
			Orchard grass negative	
			Ragweed short negative	

All other routine tests were negative, as well as wheat gliadin, corn, walnut, celery, and wool. Feathers were quite negative.

He was next seen on February 17, 1923. He had been well during the winter, but on this day he had smothered breathing and had to burn a powder. No vomiting. He was retested to horse dander, feathers, and wheat globulin. Horse dander gave +++, the others being again negative. On March 17th he was tested with horse dander dilutions: 1/500 was ++++ and 1/100,000, +. It was therefore decided to desensitize him to horse dander, and treatment was commenced on March 21st with 1/1,000,000 dilution. I append a diary of his treatment, with remarks opposite each injection, of the events following it:

1923.

March 21,	1/1,000,000, ♏	2	—No reaction; March 25th got feet wet, then had sneezing and cough and heavy breathing for two days.
" 30,	1/500,000,	1½	—Fine until A. M. April 5th, little wheezy, weather damp and cold.
April 5,	"	2½	—April 6th, asthmatic and vomited—church concert, well otherwise. (Two minims would have been enough.)
" 10,	"	3½	
" 16,	"	5	
" 23,	"	7	
" 30,	"	8	—No attacks since—quite well.
May 7,	1/100,000,	2	—Quite well.
" 14,	"	3	—Quite well.
" 21,	"	4	
" 28,	"	5	—Has had sneezing every day—nothing else. Three very slight attacks of asthma commencing May 29th. No local reaction.

1923.

June	5,	2/100,000 ♏	5 —Quite well.
"	12,	"	6 —Two attacks of asthma twenty minutes each on 15th and 16th.
"	18,	"	7
"	25,	"	8
July	3,	"	9 —Has been well.
"	10,	"	10 —Since July 4th or 5th he wakes with sneezing and heavy breathing for a short time, very mild. Inhales powder and takes rhinitis tablets for sneezing.
"	16,	1/10,000,	1½—(1½ minims of 1/10,000 represents 15 minims 1/100,000 and is too great an increase.)

A few days after this he was taken by his parents to Ontario. Asthma began on the train, and he was troubled with it most of the time until his return to Montreal about August 23d. His treatment injections were continued by the local doctor:

1923.

Aug.	27,	1/10,000, ♏	6 —Fine all week.
Sept.	4,	"	6½
"	10,	"	7½—"Cold" same day, asthma for three to four days.
"	17,	"	7 —Slight attack night of 23d for ten minutes.
"	24,	"	8 —All right.
Oct.	1,	"	9 —Attack one day for ten minutes.
"	8,	"	9 —No trouble.
"	15,	"	9 —October 23d had an attack. Missed one treatment.
"	29,	1/1000,	1½—Two slight attacks night of October 29th and next day. Local reaction plus.
Nov.	5,	"	1½—No symptoms, local plus.
"	13,	"	1½—Fine.
"	19,	"	2 —Fine.
"	26,	"	2½—Asthma night of 28th, lasting all night and next day. Began at noon on 28th with sneezing. Tongue was coated. Mother gave calomel. Eczematous patches on neck. Had headache with the sneezing (?) "Cold."
Dec.	3,	"	2½—
"	10,	"	3 —All right.
"	17,	"	4 —All right.
"	26,	"	5 —All right.

1924.			
Jan.	3,	1/1000 ♏	6 —All right.
"	10,	"	7 —All right.
"	17,	"	8 —All right.
"	24,	"	10—All right.
"	31,	1/500,	5 —All right.
Feb.	7,	"	6 —All right.
"	14,	"	7 —All right.
"	22,	"	8 —All right.
"	28,	"	9 —All right.
March	6,	"	10—Has been perfectly well, no sneezing or anything.

In going over his records I felt that there was a "nigger in the woodpile," and on March 6, 1924 he was retested with the pollens, and gave the following results:

Timothy,	+++++	Ragweed short,	++
Orchard grass,	++(+)	Sunflower,	++
June grass,	+(+)	Goldenrod,	+
Red top,	+		
		Tree pollens.	
Daisy,	++	Maple,	
Clover,	+(+)	Poplar,	
Dandelion,	+(+)	Walnut,	
Rose—negative.		Willow,	
		Elm—all negative.	

This was somewhat of a shock, as I had been lulled to sleep by the tests done in October, 1922.

Now, if we glance back over our diary of treatment, we can understand the symptoms which occurred about the middle of June, and continued at intervals during July, being worse in the country, as these were in all probability due to timothy pollen.

Ragweed probably accounted for his asthma during the latter part of August and in September; and sneezing, etc., which occurred the end of May last year may have been due to dandelion. The attack on April 6th may have been due to too big a dose on April 5th—2 minims would have been enough.

We shall, of course, desensitize him to timothy pollen.

Comment.—These cases are cited in detail to show the value of checking up on the facts, and also of repeating the tests in those cases where the results do not seem to be satisfactory.

VASOMOTOR RHINITIS

Vasomotor rhinitis is a condition which we occasionally have referred to us from the Nose and Throat Department.

Case IX.—Miss A. G., aged nineteen years, was sent to us for protein investigation on May 1, 1923.

The history was that for the last four years she had had very frequent colds, both winter and summer. These are characterized by sudden onset, blocking of the nose, later by profuse watery discharge, and an irritation or soreness inside the nostrils.

Personal history negative except for one attack of hives. She works in a hat factory.

Family History.—Maternal grandmother always sneezed when she went near flour.

Protein tests were done and positive reactions were obtained as follows:

Wheat globulin,	+(+)
Barley,	+
Pea,	+
Bean,	+
Celery,	+(+)
Tomato,	(+)

She was instructed to omit barley, peas, beans, and celery from her diet, and to restrict the use of wheat to crisp toast.

When last heard from she was almost completely free from symptoms of vasomotor rhinitis.

Case X.—Miss B. McD., was referred to us March 6, 1923.

History of Present Illness.—Two years ago she had an attack like hay-fever, which began in July or August and lasted on and off until late in the fall. Since then she has been well until about six weeks ago, when she developed a cold, with itchy eyes and nose, watery secretion from the nose, and a lot of sneezing. Sneezing is especially marked in the mornings.

Protein tests were negative except to orris root, which gave a ++ reaction, the hive 1 cm. in diameter, and erythema of $1\frac{1}{2}$ cm.

She then admitted that using face powder would generally make her sneeze and her eyes water; also some tooth powders.

She was advised to sacrifice the "dull finish" to a sense of comfort. Since giving up face and tooth powder she uses one handkerchief a day instead of six or seven, and her vasomotor rhinitis is cured.

Case XI.—Miss E. S., aged fourteen years, referred February 20, 1923 for protein investigation.

The condition began last winter, and has recurred this winter also. She has very bad colds in her head, with a great deal of sneezing and irritation in the nose, and a profuse thin watery discharge. There is also soreness in the meatus. She is perfectly free in the summers.

Personal History.—School girl. Good digestion, no hives or eczema. Appendix operation at six years. Prior to this used to have bilious attacks.

Protein Tests:

Goose feather, +
Wheat globulin, +
Orris root, +

All other proteins were negative.

When asked delicately if she ever used face powder, she replied, "No, not now, I gave it up for Lent." On further questioning it was discovered that since Lent began she had not been troubled with symptoms.

The subsequent history has seemed to bear this out, as we were content at the outset to eliminate the orris root; and nothing has been said about feathers or wheat.

Comment.—These cases represent types of perennial hayfever which may be due to face powder or to foods. Besides orris root, rice is a constituent of many of the better face powders, and is not infrequently found to be the cause.

ECZEMA

The next 2 patients which I have to show are cases of eczema. The first in a child of two years, and the second in an adult.

Case XII.—R. M., aged two years.

Personal History.—Breast fed for twelve months. When four months old it had an eczematous patch on the left cheek, which the mother thought was due to teething. It was brought to the hospital and cured.

History of Present Illness.—Three months ago the child began to have an eczematous eruption on the arms, face, and legs. The condition was itchy, and with scratching abrasions occurred, with secondary infection and formation of scabs. For the last two months the child has been attending the Outpatient Clinic, and was there tested by Dr. Williams for sensitiveness to various proteins. The only ones which were at all positive were wheat and oat. The mother was instructed to withhold wheat flour and oatmeal, but she found it impracticable to rigidly adhere to instructions. However, with even partial elimination of bread, etc., there was some improvement. Referred to the Pediatric Ward for treatment and observation, to the service of Dr. S. G. Ross.

On admission (November 20, 1923) the child is a well-nourished boy of two years. There is a wide-spread eczematous eruption involving the skin of the face, neck, arms, hands and legs, and to some extent the body.

A diet was ordered to exclude wheat flour and oatmeal, and soothing ointments applied.

On November 27th, the eruption having cleared and no fresh spots having appeared, cream of wheat was given by mistake at the evening meal, and a fresh rash on the hips and shoulders appeared two hours later. On December 6th the skin was practically clear.

December 8th, 7.30 A. M.: Cream of wheat ordered. 10.30 A. M.: Fresh rash appeared on shoulders.

December 11th: Skin was clear.

December 13th: Thin slice of well-dried toast given, with no reaction. The next day a bowl of oatmeal given, with no ill effects; the child was discharged.

Comment.—This case is unique in our experience as showing the rapidity of the eczematous reaction in a child sensitive to

wheat protein; and in illustrating the specificity of the particular protein in producing the eczema.

Case XIII.—Mr. W. M. P., came complaining of eczema February 11, 1924.

Personal History.—Age forty-three, manufacturers' agent in woolens. For some time past he has had very little handling. Never any trouble with skin before one and a half years ago. Hives as a child only; no angioneurotic edema, vasomotor rhinitis, or asthmatic symptoms.

Even as a child he used frequently to get "bilious attacks," with vomiting, headache, etc. Ever since he can remember he has had "indigestion"—often a heavy head, feeling dull, etc., occasionally accompanied by pains around the stomach, and sometimes with nausea. Ever since he was a lad he had noticed that there were things that he could not eat; the first thing was oatmeal porridge, which always made him "deadly ill." He has known for many years that nuts were bad for him (except cocoanuts); even almond flavoring in a cake would upset him. As a lad he found that he could not touch honey; just the slightest taste would cause his tongue and lips to swell and his palate to quiver.

He brings a list of foods which he can eat, of foods which are bad for him, and of foods which are very bad for him:

Can eat.	*Bad.*	*Very bad.*
Meats,	All nuts (except cocoanut),	Almonds,
Fish,	Tomatoes,	Cooked tomatoes,
Eggs,	All stone fruit,	Cornmeal,
Fowl,	Raspberries,	Honey.
All plain vegetables cooked,	Currants,	
Homemade pastries and cakes,	Raisins,	
Apples,	Bananas,	
Oranges,	Gooseberries,	
Grape-fruit.	Peaches,	
	Cucumber,	
	Lettuce,	
	Salads,	
	Porridge,	
	Lobster.	

Family History.—His father could not take milk in any quantities, and he had "acute indigestion" nearly all his life. He is married and has 5 children—fourteen years to three months. With all 5 children there has been difficulty with their feeding during the first two years—indigestion, vomiting, etc. One boy of five years may be eating his ordinary food, such as potato, milk pudding, or bread, and will suddenly have to leave the table to be sick. His wife says that the children have the "Potter stomach." Several members of the family have the "Potter stomach."

History of Present Illness.—The condition began on the right hand, between the first finger and thumb, one and a half years ago. It was dry, scaly, and itchy. After a month or two it came on the first and second fingers.

Then it appeared on the soles of the feet in the form of water-blisters under the skin, which he pricked with a needle. This lasted six months or more, being on one foot or the other, sometimes on both, but not itchy. Then it disappeared, but again recently it has reappeared on one sole in the same form.

In the hand it has tended to persist, being worse at times. Last summer it was severe in both hands and feet.

About three months ago it appeared as tiny itchy pimples on the calves. These were very itchy, and by scratching some of them got infected. The pimples tended to break and exude. Some boils have formed. Ointments prescribed have made practically no difference.

This patient is interesting as illustrating an extraordinary degree of multiple desensitization. Intelligent and observant, he has found out for himself many things which he dare not eat. The protein tests, which have been clear cut, have for the most part corroborated his own findings. (See page 1738.)

At first glance the outlook from a food viewpoint looks well-nigh hopeless. It is however possible to choose a diet which will exclude the positive foods; and we hope that the sacrifice may bring about an amelioration of his condition. It may prove advisable to attempt non-specific desensitization by peptone administration, as Auld has advised.

Protein Tests:

Grains and vegetables.		*Milk, eggs, meat, and fish.*		*Fruits, nuts, etc.*	
Wheat,	++	Lactalbumin,	(+)	Walnut,	+++
Corn,	++	Casein	negative	Almond,	++(+)
Oat,	+	Egg-white	negative	Peaches,	+(+)
Rye,	+	Egg-yolk	negative	Coffee,	++(+)
Barley,	+++	Chicken	negative	Grape-fruit,	+
Rice,	(+)	Lamb	negative	Orange	negative
		Pork	negative	Tea,	(+)
Peas,	+++(+)	Beef	negative	Cocoa,	++
Bean,	+	Mackerel	negative	Banana,	+
Celery,	++	Haddock	negative		
Potato	negative	Codfish	negative		
Asparagus,	++	Salmon	negative		
Tomato,	+++				
Onion,	++				
Cucumber	negative				
Lettuce,	(+)				

Epidermals.	
Goose feather	negative
Chicken feather	negative
Horse danger	negative
Dog hair	negative
Cat hair	negative

At the present time he presents a problem yet unsolved.

I am greatly indebted to Dr. Chandler Walker, of Boston, for the inspiration and help which I have received from him on more than one occasion when I have had the pleasure of visiting his clinic at the Peter Bent Brigham Hospital, and I would like to take this opportunity to express my appreciation.

CLINIC OF DR. ALTON GOLDBLOOM

CHILDREN'S MEMORIAL HOSPITAL

HYPERTROPHIC STENOSIS OF THE PYLORUS

GENTLEMEN: The case which I am to present to you today is one in which the chief interest lies in the fact that it presents all of the classical signs and symptoms of the condition which I wish to discuss with you. The condition itself is really a surgical one, but it is a singular fact that most surgical conditions in infants present themselves to the pediatrist first of all, and it is, therefore, his duty to be thoroughly familiar with the signs and symptoms of many surgical conditions, and at times to be able to exercise good surgical judgment; for often the very life of such a child may depend upon the pediatrist, who must know exactly when the assistance of the surgeon is required. One of these conditons, for instance, is intussusception, which, while it is purely surgical, and relieved only by operation, is scarcely ever first seen by one other than the pediatrist or internist. Accurate and quick diagnosis and early operation are, of course, essential to the life of the child in such a case. Our present case, as we shall see, is one which required the most careful consideration both of the physician and the surgeon.

The history of this case, in brief, is as follows:

The patient is a male child of healthy parents. He is the first child. Pregnancy and labor were normal. The weight at birth was 7 pounds. He appeared to be a normal infant in every way for the first three weeks of his life. He had the usual postnatal loss of weight, and then began to gain at a fairly regular rate; he regained his birth weight on the twelfth day and went on nicely until the twenty-first day. The stools were good and there was no vomiting. About the eighteenth day it was ob-

served that the child regurgitated some food after each nursing. The nursings were given every three hours, seven feedings in the twenty-four hours. The child was kept at the breast for about twenty minutes. The mother had a moderately good supply of milk. By the twentieth day the vomiting took a very definite form, which the mother described in her own words as "explosive." This occurred after the nursing, sometimes in the midst of the nursing. Apparently all that the child had taken came up with great force; at times coming through the infant's nose, always shooting out a great distance, that is, 2 or 3 feet from the baby. You know that this is not characteristic of any type of vomiting with which you are familiar in infants. Infants may vomit from excessive feeding; from excessive fat in the mother's milk. Infants may vomit by being fed too often, which is practically the same thing as being fed too much, but the vomiting is never such that practically all that the infant takes comes up immediately after it is nursed—and sometimes during the nursing; sometimes even with evidences of pain immediately before the vomiting starts. Such was the case here. By the twenty-first day the child was vomiting after every feeding. Coincidently with the vomiting it became extremely constipated, and the urine, which is usually very abundant in a young infant, occurring almost every hour, became so scanty that the child urinated only once in about seven or eight hours, and even then only a small quantity, which was so concentrated that it left a brown stain on the diaper. This was allowed to go on for four or five days before medical advice was sought, and the physican who was called came to the conclusion that the mother's milk was disagreeing with the baby, and he promptly proposed weaning. The child was weaned and was put on a simple milk and water dilution with the addition of lactose. It was fed every three hours and the vomiting persisted. It was fed in very small quantities every two hours, still without effect; it was fed every four hours, still without effect; still with excessive emaciation and with continued constipation and with scanty urine. It was at this time that the child was admitted to the hospital—six and one-half weeks old.

The best weight had been $8\frac{3}{4}$ pounds at twenty-one days. It was on admission 6 pounds, 12 ounces, having lost nearly 2 pounds in the first week of its illness.

When first seen, after admission to the hospital, the child was an emaciated infant without any subcutaneous fat, alert, always looking for food, apparently always hungry. He was obstinately constipated, so much so that an enema was required in order to have the bowels move at all, and it looked as if the child were in imminent danger of dying.

We will confine our remarks concerning the physical examination to the abdomen. The abdomen was rather full in the upper half and rather retracted in the lower half. The outline of the stomach could be seen. When the child was given a little water in a bottle it was observed that there were peristaltic waves visible in the left upper quadrant of the abdomen, commencing just below the ribs and traveling from left to right toward the navel, disappearing in the region of the navel. On palpation of the abdomen a small movable mass, about the size and shape of the distal phalanx of my little finger (hard as cartilage and movable) could, on careful palpation, be elicited in the right upper quadrant 1 inch above and 1 inch to the right of the navel.

The question is, What are we dealing with? and I think that it is not necessary to go very much further into the case to at least suspect that we are dealing with a stenosis somewhere in the gastro-intestinal tract. We are dealing with an obstruction; we have got the signs of obstruction, though not the signs of complete obstruction. There is some food getting through the intestines, because the child, when urged by means of laxatives or by means of enemata, will have a stool, and the stool will show some signs of food having been digested; at the same time it is very small in quantity; there is vomiting and there is emaciation.

The vomiting takes place immediately after food is given; waves of peristalsis are visible, and very plainly visible, in the left upper quadrant of the abdomen. Waves of peristalsis are visible in the region of the stomach and travel in the direction

in which gastric peristalsis travels, namely, from left to right; the rest of the abdomen is collapsed, and I think we must assume that if there is an obstruction, or a partial obstruction, that obstruction is probably somewhere between the stomach and the rest of the intestinal tract. We are able to feel a small, hard tumor in the right upper quadrant of the abdomen, and I think we can almost reasonably assume that this is a thick, hypertrophied pyloric muscle which probably has become so hypertrophied as to almost completely obstruct the lumen of the intestinal canal at that point.

There are one or two things that we can do to prove that we are dealing with a case of hypertrophic stenosis of the pylorus. One of these things is to use the *x*-ray. We could do this but for the fact that it is a great hardship to an infant who is as emaciated as the one which I have just described for you. Further, if we did give this child a barium meal, I am sure it would show a prolonged retention in the stomach. It is not necessary, however, to take the time or subject the child to the inconvenience of the *x*-ray, when we have a very simple device for seeing whether the child is passing any food, or any considerable quantity of food, through the pylorus or not. We will give a measured quantity of food which we will allow the child to take, and at the end of three hours we can pass a catheter into the stomach and by means of a syringe withdraw the gastric contents. I believe we can assume that if more than two-thirds of the food given has left the stomach at the end of three hours the obstruction is not at the pylorus. If, however, we recover more than two-thirds of the amount given or, better still, if we recover more than we gave, we can be perfectly sure that an obstruction at the outlet of the stomach exists if we can rule out slow or delayed gastric motility. In this instance we have ruled it out by the discovery of forcible visible waves of gastric peristalsis. That is really a hypermotility of the stomach which immediately throws out the possibility of delayed motility.

In this instance we gave 60 c.c. of breast milk (breast milk being the physiologic food for the infant of that age) and, therefore, we would assume that with breast milk the emptying time

of the stomach would be what it normally should be. (The emptying time of the stomach is not always the same with all foods.) Breast milk being the physiologic food, it was given in a small quantity, 60 c.c., and this was given immediately after a gastric lavage, so as to be sure that the stomach was empty at the beginning of the test.

The child did not vomit the 60 c.c. of breast milk, but at the end of three hours 50 c.c. of fluid was withdrawn from the stomach, which I believe proves to us that we were dealing here with a very typical instance of congenital hypertrophic stenosis of the pylorus.

Now let us go back a moment and consider all the points which have led us to this diagnosis.

In the first place, we have a male child seven weeks old. Has the sex any relation to the diagnosis? It has to this extent—that about 1 in 12 or 1 in 13 only of cases of hypertrophic stenosis of the pylorus occur in girls; the overwhelming majority occur in boys. Next, it is the first-born; it occurs in other children as well, but about half of all the cases reported are in first-born infants.

So then we have a boy, first-born, with the outstanding symptom vomiting, and the vomiting was of a very characteristic type, it was not mere regurgitation, it was not a mere overflow vomiting, such as is very commonly seen in children who are being overfed or who are fed too frequently (which amounts to practically the same thing), and which is of no clinical significance (for instance, children may sometimes gain, and gain beautifully, while doing that type of vomiting); we have the kind of vomiting which occurs immediately after each feeding, which comes up with great force, the child certainly vomiting nearly as much as it has taken at the feeding, leaving the child hungry immediately after it had vomited (not so, of course, as in vomiting which is due to overfeeding, when the child merely gets rid of an excess, leaving him satisfied), followed by emaciation, constipation, and small, infrequent quantities of urine, highly concentrated, so concentrated, in fact, as to stain the diaper, the urine occurring only two or three times

in the twenty-four hours. We have the waves of peristalsis which are perfectly typical of gastric peristalsis and typical of nothing else, and we have the palpable mass in the right upper quadrant which is caused by the pyloric thickening.

Before we discuss the treatment let us discuss for a moment the pathology of this disease.

Is it a congenital condition? You would say to me, If this is a congenital condition, how is it that the child was perfectly well for three or four weeks before any symptoms developed? For answer to this we may say that the condition has been seen in newborn infants, and has been reported as showing well-marked signs as early as the second or third day after birth, and a certain percentage of infants have vomited from birth. In those that have not vomited from birth, but in whom the vomiting commenced only two or three or four weeks after birth, prior to which the child was apparently well, we assume that the hypertrophy of the pyloric muscle was present from birth, and we argue that something more than a mere hypertrophy of the pyloric muscle is required to cause symptoms. Added to the hypertrophy of the pyloric muscle we have probably a physiologic derangement of the neuromuscular supply to the pylorus. This has been suggested by some anatomists —that the mechanism which opens and closes the pylorus at the proper time is deranged, either coincidentally with or as a result of, the hypertrophy of the pyloric muscle, and that it is this derangement of co-ordination between gastric contraction and pyloric contraction that causes the symptoms which I have described. Witness, for instance, that cases of pyloric stenosis can be divided into two large groups—mild and severe. Cases may vary from ones that are so mild as to recover spontaneously without any treatment, to ones in the severe group which are so severe that they vomit from the day of birth, lose weight rapidly, and very early show signs of almost complete obstruction. In some of the mild cases it is a question sometimes of nicety of opinion, whether medical or surgical treatment is preferable, and when such a child comes to operation it is observed that there is no relationship whatever between the size

of the pyloric tumor and the severity of the symptoms. The same has been noted in very severe cases. There is always some hypertrophy of the pyloric muscle, but there may be a very large tumor in a case showing the mildest possible symptoms, and there may be only a very moderate hypertrophy in a case showing the most severe symptoms. So we have more than the mere mechanical thickening of the pylorus, we have the hypertrophy of the circular muscular coat which puckers up the mucosa of the pylorus to a considerable degree, added to which is the element of spasm which completely obliterates the pyloric orifice, and which, I think, is mostly responsible for the symptoms which I have just described.

How are we going to treat such a case? We have an infant with symptoms of intestinal obstruction, not complete. It is obvious, I think, that in this particular case the only treatment which can be of avail is a treatment which will render the infant's pylorus permeable to food at the quickest possible moment. We have not an infant here with whom we can waste four or five days trying to see whether this food will do it, or whether that food will do it; or trying to find out whether thick food will pass through the pylorus, or whether giving atropin will relieve the spasm, or trying to find out whether larger or smaller quantities of food at frequent or infrequent intervals will pass through; we are dealing with a surgical emergency, almost as much of an emergency as an intussusception or a strangulated hernia. True, we have not a complete obstruction, but we have an obstruction which is so great as to prevent the child from receiving into its intestinal canal sufficient food and sufficient water to maintain the ordinary metabolic functions, to say nothing of providing for growth or for activity. We have a mucosa which is puckered up so as to be almost completely obliterated, and which allows a minimum amount of food to pass through; we have a very thick inner circular muscular coat of the pylorus (the muscular coat in an instance of this type is sometimes 3 to 5 mm. thick), and which goes into periodic spasm causing intermittent complete obstruction. We have a child who is very rapidly losing ground, and we must get food

into its intestinal tract as quickly as possible in order that he may have a chance for life. What must we do? We must incise the circular muscular fibers of the pylorus; we do not have to incise the mucosa; we have to divulge the edges of the incision so as to allow the imprisoned mucosa more freedom, and I think in doing that we do one other thing, and that is, we make it impossible for the pylorus to contract down over the mucosa when it goes into spasm.

We do this nowadays as a result of some work that was done as far back as 1908 by Fredet, and a little later, in 1912, by Rammstedt. Prior to the popularization of a method which they both independently evolved, the method of operation was gastro-enterosteotomy, and you all know how serious an enterosteotomy operation is in an adult, and how much more serious it would be in an infant, who is not only of tender age, but who has suffered from the most severe effects of malnutrition, and, therefore, is in no condition to stand a prolonged operation.

Following the falling of gastro-enterosteotomy into disrepute for relief of this condition, the Fredet-Rammstedt operation has become so popular that this, whenever operation is done, is used to the exclusion of any other form of surgical therapy.

The operation is so simple that it can be done in the space of ten minutes. A short, longitudinal incision is made in the right upper quadrant of the abdomen through the skin and rectus muscles. The peritoneum is incised and the pylorus immediately delivered into the wound; the surgeon then cuts longitudinally, the incision extending from the end of the stomach to the end of the pylorus, altogether about 1 to $1\frac{1}{2}$ inches, and then with blunt forceps divulges the edges of the incision until he can see the freed mucosa. The cut edges of the pylorus can be so spread that the mucosa is visible throughout the whole length of the incision. Ligatures are hardly ever necessary. Bleeding is controlled by hot pads. The pylorus is dropped back into place, the wound is sewn over, and that is all there is to do—a very simple operation indeed.

This was done in this case. The anesthesia was ether. The operation lasted twelve minutes. At the end of the operation the child was in moderately good condition, although he weighed but $6\frac{3}{4}$ pounds at the end of seven weeks, and weighed $7\frac{1}{2}$ pounds when he was born. He stood the operation well. Immediately after leaving the operating-room he was given a subcutaneous saline, which was absorbed very quickly; he was given water by mouth as soon as he was able to take it, and food was commenced within a very short time after his recovery from the anesthesia; that food was human milk, and was given in very small quantities at first—as small as a teaspoonful every two hours, gradually increased, until at the end of twenty-four hours he was receiving 1 ounce every three hours. At first it was slightly diluted, later on, at the end of a day, it was not diluted, and pure breast milk was given.

There was no more vomiting after the operation. The child did very well, and began to gain about the eighth day after operation. The food was gradually increased, and the sixth day after operation he was put to the breast. At the end of ten days he was discharged from the hospital and went home perfectly well, nursing his mother.

Now, this baby had been weaned; he had been off the breast for about three and a half weeks. In the first place, it is a grim commentary on weaning a baby whose mother has abundant milk, on the assumption that the milk is disagreeing with the baby. I often wonder upon what grounds we have the right to assume that the milk of a healthy mother cannot agree with her own baby—why blame the mother, why not blame some radical derangement in the infant? One should certainly weigh the evidence, and not assume that it is the mother who is at fault when you know that there are two possibilities. We are too prone to jump to the conclusion that the mother's milk is at fault, but we never jump to the conclusion that the baby is at fault, and I think that both possibilities must be given equal consideration; and if equal consideration had been given to both possibilities in this case the infant would never have been weaned.

There was nothing wrong with the mother's milk. Fortunately, three and a half weeks is not sufficient length of time to completely drive away the secretion of the breast. I have seen abundant secretion in the breast of a woman whose baby has been weaned twelve weeks, and I have put that baby successfully back on the mother's breast at the end of twelve weeks and have re-established her milk supply. I have had similar success with infants who have been off the breast for six weeks and four weeks respectively; therefore, we did not consider it a very great task to re-establish the milk of this mother, whose infant had been away from the breast for three and a half weeks. There was abundant secretion in the breasts, and it only required a little stimulation to bring it back. Her own infant was too feeble to nurse, and we did not think it was right to subject him to the exertion of sucking at the breast for fifteen to twenty minutes, so what we did was to give the mother a healthy, vigorous infant to nurse her breasts, and at the same time we made her drink abundantly of fluids and eat well. It was surprising how quickly her breasts were as full as those of women three or four days after parturition.

So the baby was put back to the breast, and everything went very nicely.

Hypertrophic stenosis of the pylorus belongs to the physician because it is going to come to the physician rather than go to the surgeon, and it depends upon the physician's acuteness of diagnosis whether a child's life will be forfeit to his ignorance or saved by his ability. It is of the greatest importance that every man who does general practice, or who does anything which has to do with children, be able to recognize and recognize immediately hypertrophic stenosis of the pylorus. It is true that it is turned over to the surgeon for treatment, but the length of time that such a child is turned over to the surgeon is merely for the duration of the operation itself, and no more. In other words, the surgeon is called in to do a certain piece of work, and once his work has been done, namely, once he has opened up the door that has been closed, the case immediately reverts back to the physician, because it requires skill

and judgment in infant feeding to carry such a case on. What I mean is, that the surgeon's duty is merely the opening of the pylorus, the physician's duty is, in the first place, to diagnose the case beforehand, suggest operation and not temporize too long, and treat the case after it has been operated upon.

Medical treatment may be permitted in rare cases, that is, in cases which are very mild, in cases which have lost but little if any weight, and in which you feel that a delay of three or four days is justifiable, to see if the child cannot be made by one method or another to retain food, but we must be extremely careful what cases we choose as medical cases. Anyone who has seen any number of cases of pyloric stenosis at all has seen cases which have been sacrificed to a whim at a time when the well-tried method of merely incising the pyloric muscle would have saved the lives of these infants.

If, on the other hand, a perfectly good case of well-marked pyloric stenosis is seen in a child who weighs 9 or 10 pounds, who is six or eight weeks old, and who is not completely obstructed, but only partially so, and who is losing very slowly, I think one's judgment would be correct if one tried one of the recommended methods of treatment.

Now what are they?

One method of treatment suggested by Howland has been the method of refeeding. Refeeding means that the infant, immediately after it has vomited, is given a second feeding of the same food, and if it vomits the second, it is then given a third feeding immediately after having vomited the second feeding; the idea being that the spasm is relieved immediately after the attack of vomiting, and that the food, after a second or third feeding, may be retained.

Another method that has been recommended has been the thick cereal method of Sauer, the rationale of which is to give a food which is so thick that it cannot be vomited. Therefore, if it remains in the stomach a sufficiently long time, it may be there at a time when the pyloric spasm is temporarily relieved. We have recently seen a child with a mild case of pyloric stenosis who did well on just this method of feeding. When

first seen it was four months old, and had maintained a stationary weight almost since birth. The vomiting occurred two or three times each day, and was accompanied by the visible gastric peristalsis and there was palpable tumor. At the same time the stools, though they were somewhat constipated, contained a certain amount of digested food; they were not the typical starvation stools which are seen in the marked cases. It was our opinion that since this child had lived for four months with practically no loss of weight, he had a good chance of recovery if the thick cereal method were adopted. A gruel was made with skimmed milk and farina which was cooked for about two hours until it was quite thick. This was divided into five equal portions, and one portion was fed by spoon every four hours. Water was allowed between feedings. This child stopped vomiting immediately and began to gain. After two months on this treatment he had gained 5 pounds.

Another method is the atropin method suggested by Hass, which consists of giving atropin in fairly large doses, starting with 1/1000 grain immediately before each feeding, and working up to sometimes as high as 1/250 grain immediately before each feeding. It is sometimes accompanied by flushing, but by no more severe symptoms. Results from this method of treatment have in my experience been variable. But my impression is that atropin may be more useful in conjunction with one of the other methods mentioned, especially the thick cereal method, than it would be when used alone as the sole method of treatment.

My personal bias is in favor of the thick cereal method if ever medical treatment is to be tried. And let me warn you once more that medical treatment is to be tried only after careful consideration and only in a case which is not losing weight too rapidly. Whether you try medical treatment or not, there is one fact I want to impress upon you, and that is that there is no method of treatment of hypertrophic stenosis of the pylorus which is as economical as surgical treatment. Medical treatment is time consuming; progress is extremely slow; and if the child is in the hospital, the length of time the child must stay

in the hospital is far longer than is good for a child of tender weeks. Cases that have been operated on are usually at home and thriving within ten days after the operation. The cases treated medically are generally in the hospital for a month or two or sometimes longer.

A word about the prognosis in pyloric stenosis. The prognosis in hypertrophic stenosis of the pylorus depends upon the rapidity of the weight loss, the severity of the symptoms, whether or not the child has been weaned before operation, and the length of time delayed before operation is performed.

In a group of cases reported by Spence and myself a number of years ago we found, for instance, that the mortality was as low as 5 per cent. in the cases in which the operation was performed when the symptoms had lasted only a week or less, but when the symptoms had lasted as long as five weeks or longer the mortality was 40 per cent. If the infants were breast fed at the time they came to operation, that is, if they had not been weaned, or if they could readily be put back on the breast, the mortality was as low as 11 per cent., and if they had been weaned before coming to operation the mortality was as high as 35 per cent. In those that weighed 5 pounds or less at the time of operation the mortality was 40 per cent.; while in a series of 6 cases in which the children weighed 9 pounds or more at the time that they came to operation, there was no mortality.

Out of a group of 94 cases that weighed less than 7 pounds at the time of operation, the mortality was 28 per cent., and out of a group of 69 cases that weighed more than 7 pounds at the time of operation, the mortality was only 8 per cent.

Among the infants who were operated upon before they had not lost more than 10 per cent. of their best weight, there was no mortality at all, they all recovered; and the mortality mounted as the weight loss mounted; the greater the weight loss, the greater the mortality, until it reached 50 per cent., when more than 30 per cent. of the best weight had been lost.

These figures argue, of course, that if cases of pyloric stenosis are definitely diagnosed, if they are losing weight rapidly, and if they show all the classical signs which I have outlined, the

quicker the operation is performed, the surer you are of a successful issue.

If, however, we are dealing with an instance where the symptoms had been moderately mild and the weight loss not too severe, it is permissible to try some form of medical treatment for a short time, and if immediate and gratifying results are not obtained, to temporize no longer, and in these cases the mortality from operation in pyloric stenosis will be no greater than the mortality from, say, an interval appendix. There is no reason why all practitioners should not recognize pyloric stenosis immediately, or if not when first seen, certainly within a day or two. There is no reason why it should not be suspected in every child where the vomiting is persistent, recurs after every feeding, is of an explosive nature, and especially when accompanied by constipation and an associated infrequency of urine, and if you want a conclusive test have a barium *x*-ray taken of the stomach, but by all means remember that these cases, as soon as they are diagnosed, if they are severe, should be operated upon at once; if they are mild they may be temporized with, but not for too long. Do not make too many changes in the food; plan out a definite well-set plan for medical treatment, if you choose the medical treatment, follow it for a few days, and if you find that you have not been successful, refer it to the surgeon immediately.

CLINIC OF DR. I. M. RABINOWITCH

MONTREAL GENERAL HOSPITAL

ON CERTAIN PROBLEMS IN THE TREATMENT OF DIABETES MELLITUS

GENTLEMEN: In this clinic I wish to discuss certain problems not infrequently met with in the treatment of the diabetic. Sir J. Rose Bradford put it very well when he stated that "diabetes is not an entity, but a clinical label attached to a number of different conditions, with varied origin, different morbid anatomy, and liable to follow different courses." The truth of this statement becomes only too evident in the course of the treatment of a series of patients.

It may be stated, in a general way, that in the treatment of diabetes there are now either one of two courses to follow—treatment with or without insulin. With very few exceptions the better course to follow is fairly well defined by a careful study of the clinical and laboratory data combined. Since the advent of insulin it appears that there has been a tendency —as there always is with the introduction of any new therapeutic agent—to apply the remedy very freely. Before the discovery of insulin Allen demonstrated, by dietetic measures, that, with very few exceptions, a stage can be reached which is represented by the intersection of the curves of falling weight and rising tolerance, at which life can be maintained. This stage should be determined by means of all available laboratory facilities before insulin is employed. If at this stage the patient must be kept in a state of nutrition incompatible with the normal functions of the individual, insulin is then indicated. Experience in this hospital has taught us that the determination of this stage has been of value not only as an index for

insulin treatment, but especially from one other point of view, namely, that of prognosis. An important question is whether a diabetic who once requires insulin can subsequently do without it, diet alone sufficing.

In this discussion we are excluding one type of case, that in which a sudden severe derangement of the pancreatic function has occurred, superimposed upon an existing mild diabetes. Such a derangement might be due to trauma, infection, onset of the menstrual epoch, etc. Such patients, though even progressing to the stage of coma or actually in coma, may by the use of insulin recover to the stage of diabetes previously existing. Though for a time such patients require enormous amounts of insulin, 75 to 100 units or more in a day, following the elimination of the exciting factor, they acquire an enormous tolerance, and subsequently do well without insulin. This is not the type of patient we are to consider now.

We are now concerned only with the well-recognized type of chronic diabetic, who though steadily progressing downward, this progress is not due to any such recognized factors as those referred to above. With this type of case—as far as can be judged from the short period during which insulin has been used—the experience in this hospital has been that such a diabetic, actually requiring insulin, is subsequently unable to do without it. An analysis of the data from patients who have been able to return to dietetic measures alone, that is, whose tolerance has apparently improved under insulin treatment, shows that they would have progressed favorably without insulin.

Diet thus appears to be a more important problem than ever before in the treatment of diabetes, and it is the purpose in this clinic to discuss certain phases of this problem.

It may be stated, generally, that there are two types of dietetic treatment: (*a*) That based upon the principle of undernutrition, and (*b*) that based upon the principle of supplying the total basal requirements, so-called basal diets. Because of the objection by most patients to the essential part of the Allen treatment, namely, starvation, numerous modifications

have been attempted. Chief among these has been the attempt to maintain the body weight by replacing with food the energy expended under basal metabolism conditions. Various formulæ for the calculation of such diets have appeared from time to time. With the availability of modern laboratory facilities such a procedure appears, theoretically, to be comparatively simple. The total energy expended by the patient is either experimentally determined or actually calculated, and the carbohydrate, fat, and protein constituents of the food apportioned to maintain nitrogen equilibrium produce a proper antiketogenic balance, and keep the patient's weight constant. In actual experience, in a statistical sense, in a large series of cases these methods hold, but in certain individual cases all these methods fail.

Let us now consider the possible causes of such failures. In the evaluation of any therapeutic measure clinical experience must be the final court of appeal. As in the physical sciences, if the deductions from an algebraic equation do not correspond to experience, either the reasoning is wrong or certain unrecognized variables enter into the problem, and have, therefore, been neglected.

When we compute a diet by any one of the formulæ in use we assume that we know:

(*a*) The chemical composition of the food,

(*b*) The ketogenic and antiketogenic values of the food, and

(*c*) That using the body weight and nitrogen equilibrium as a guide, the food ingested is the food oxidized without alteration in amount or proportion of the different constituents.

As a matter of fact, these assumptions are only approximately correct. The careful chemical analysis of food essential for accurate metabolism experiments cannot be employed for practical purposes. Here we must accept the average values of the chemical composition of food materials. A glance at the Atwater-Bryant Food Tables shows the wide discrepancy which may be found between the maximum, minimum, and the average values of the common foods. All the formulæ are based upon the assumption of a definite antiketogenic ratio. Yet the true

ketogenic value of protein and antiketogenic value of fat are still imperfectly understood. The experiments of Richardson demonstrate the fact that constancy of body weight must not be accepted, uncritically, as evidence that the food oxidized is the same in quantity and proportions as the food ingested. Another factor is the changes in body weight due to fluids. This applies especially to severe diabetics where the changes in the fluid content of the body are most strikingly demonstrated. With no restriction in salt and water intake, diabetics in a state of undernutrition have been frequently noted to gain or lose 3 to 6 pounds in a day. Therefore, where we accept these assumptions as correct, these formulæ for computing diets convey an erroneous impression, namely, the extreme degree of accuracy with which diets can be calculated.

Though such factors as I have discussed may account for a great part of the discrepancy between theory and experience in some cases, the greater part, at least in our experience, is due to another factor, namely, that of overfeeding, though this is not theoretically apparent from the calculations of the basal diets. For some unrecognized reason certain diabetics maintain and actually gain weight upon diets, theoretically, below their calculated requirements. Numerous cases have demonstrated this point, and one cannot help but conclude that dietetic treatment based upon the principle of undernutrition has yielded better ultimate results than that based upon basal diets. These observations apply equally to the treatment of those cases which require insulin. The results obtained with insulin where combined with a diet in the nature of a minimum requirement, rather than with a tendency toward overfeeding, have at least so far yielded better results. It appears that such results are to be expected. Allen has demonstrated that undernutrition should be continued to the point of relieving the pancreatic function from overstrain. Lack of thoroughness in relieving the pancreatic function is the chief cause of the deterioration of this function.

I wish now to put out a new problem we have met with in a few cases during the course of treatment with insulin. The

presence or absence of glycosuria, under ordinary conditions a recognized reliable index of progress, ceases to be such in certain cases under insulin treatment. In these patients there may be found quite a marked hyperglycemia, the blood-sugars ranging from 0.2 to 0.3 per cent., with no glycosuria. Accompanying this hyperglycemia a polyuria may also be present. Clinically, these patients appear very well, maintaining or actually gaining weight. This apparently raised threshold differs from that found at times in chronic diabetics in that it may appear within a short period of time; at times noted within a few days after the beginning of treatment, and is not associated with impairment in renal function. The case recorded here demonstrates this phenomenon. The past history is briefly as follows:

Case I.—6742, B. M., aged thirteen, was admitted to the Montreal General Hospital on December 2, 1922 practically in coma. Under insulin treatment she recovered, and on December 21, 1922 was discharged on a diet of 2044 calories. The blood and urinary findings were then normal. No insulin was further required. The diabetes at that time appeared to be of a mild type, but aggravated temporarily by an acute infection which brought on coma. Blood analyses made every two weeks thereafter while on this diet were persistently normal until May 19, 1923, when she developed a mild respiratory infection, not unlike influenza in nature. Since then glycosuria has been a frequent phenomenon, and beginning July 31, 1923 insulin was given in small doses.

Because of the unsatisfactory progress of the patient thereafter at home she was readmitted for observation on December 26, 1923. Until January 10, 1924 there appears nothing peculiar about the data, and for this reason these are not recorded. The threshold level appeared to be that of the average individual. At a blood-sugar of 0.175 per cent. glycosuria was noted. In the table are recorded the essential data from that date to demonstrate this phenomenon.

Urine.					Blood.		
Date.	Volume.	Sugar, per cent. grams.	Acetone.	Tit. acid + NH_3 c.c. N—10	NH_3.	Sugar, per cent.	Cholesterol, per cent.
January							
10.....	2700	0	0	842	1.06	.217	.227
11.....	2800	0	0	739	0.91		
12.....	2600	0	0	728	0.88		
13.....	2800	0	v.f.t.	795	0.99	.232	.223
14.....	2400	0	0	778	0.88		
15.....	3000	0	0	816	0.97	.217	
16.....	2700	0	0	799	0.97		
17.....	2160	0	0	803	0.93		
18.....	2360	0	0	...	....	.244	
19.....	3000	0	0	744	1.02		
20.....	2700	0	0	875	1.01	.212	
21.....	3300	0	0	972	.55		

The polyuria and hyperglycemia in the absence of glycosuria is here shown. There was no evidence of an acidosis. Clinically, the patient feels and looks very well and is gradually gaining weight.

Case II.—In one case showing this phenomenon the effect of increasing the dose of insulin was noted, and the essential data obtained are recorded in the table on page 1759.

Here again there is noted a hyperglycemia and polyuria in the absence of glycosuria. The data are recorded from the date the dose of insulin was increased. In this case the attempt to reduce the blood-sugar values was finally successful, but it will be noted that the polyuria has persisted. Clinically, this patient also feels and looks well.

My object was to point out the possible causes of failure at times of certain dietetic measures, and obviously not to discount the value of calculating diets, because, as Richardson points out, "without a weighed diet the physician may be totally at sea; with it, he needs only to apply the mental procedures, which he uses in every diagnosis, that is, the effort to visualize the processes which are going on within the tissues of the patient."

Date.	Volume.	Sugar.	Acetone.	Blood-sugar, per cent.
November				
10.....	3400	0	0	0.227
11.....	3260	0	0	
12.....	2730	0	0	
13.....	2600	0	0	
14.....	2800	0	0	0.192
15.....	2600	0	0	
16.....	2800	0	0	0.200
17.....				
18.....	3380	0	0	
19.....	2740	0	0	0.178
20.....	3100	0	0	
21.....	3280	0	0	
22.....	3520	0	0	0.180
23.....	3560	0	0	
24.....	....	0	0	
25.....	2440	0	0	0.188
26.....	2220	0	0	
27.....	2020	0	0	
28.....	3000	0	0	0.135

This hyperglycemia and polyuria in the absence of glycosuria noted in a few cases treated with insulin must undoubtedly cause some anxiety. Such patients, though aglycosuric, are exposed to the various complications of diabetes. One patient under observation developed a carbuncle. The disturbing feature is that the patient after being discharged from the hospital has no means of detecting any change in his progress in the absence of glycosuria.

CLINIC OF DR. EDWARD H. MASON

ROYAL VICTORIA HOSPITAL

INSULIN, CARBOHYDRATE TOLERANCE, AND WEIGHT

ONE of the questions uppermost in the minds of the men working with insulin today is whether through its use a definite improvement in the carbohydrate tolerance of the patient can be realized. That is an improvement which is not spontaneous on the part of the patient, so commonly seen in all mild and even severe diabetic cases, but one referable to insulin therapy.

This question will have to be decided through the study of severe cases of diabetes mellitus which have been given every chance to regain their impaired pancreatic function by dietetic means alone. Fortunately in this clinic we have cases of this type, and to appreciate the point involved a review of a few records will throw light on the question.

Case I.—Case No. 31,255, female, aged twelve years. Admitted November 1, 1919. Onset of symptoms was in August, 1919, with thirst, frequency, and fatigue. Glycosuria was found in September, 1919. Physical examination revealed a normally developed young girl. Wassermann was negative.

Chart I (p. 1762) shows the long downhill course that the girl experienced after discharge from the hospital in January, 1920. Living upon a weighed subcaloric diet, losing weight, not growing in height, but remaining free from glycosuria for almost three years, there seemed to be no spontaneous improvement in carbohydrate tolerance. With readmission in January, 1923 the basal metabolism was 24 per cent. below normal. Reestablished upon a diet giving 1463 calories the patient started to gain weight, to date, 22.9 kilos. She has grown 2 inches in

CHART I

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C. H. O., gm.	Total calories.	Total glucose, gm.					
1919.										
Nov. 2	45	48	30	754	60.9	30.4	65.6	0.29	0	Admission
1920.										
Jan. 26	60	35	35	715	73.3	24.4	0	0.12	0	Discharged.
Apr. 1	70	35	35	756	79.1	27.2	0	0.14	0	At home.
Dec. 1	60	35	35	715	73.3	28.0	0	0.14	0	At home.
1921.										
Aug. 23	70	60	35	989	81.6	26.6	2.9	0.18	0	At home.
1922.										
Aug. 31	70	50	30	875	75.6	26.0	0	0.15	0	At home.
1923.										
Jan. 12	60	80	50	1195	92.8	24.4	14.2	0.205	0	Readmitted.
Feb. 8	70	100	60	1463	110.6	26.8	0	0.18	46	
Feb. 15	70	100	60	1463	110.6	27.8	0	0.13	30	
Feb. 19	70	100	60	1463	110.6	27.9	0	0.14	20	
Mar. 11	70	100	60	1463	110.6	28.8	0	0.17	24	Discharged.
July 1	70	100	60	1463	110.6	....	0	....	24	At home.
Sept. 10	70	100	60	1463	110.6	39.5	0	0.17	24	At home.
Nov. 4	70	100	60	1463	110.6	42.7	0	0.19	24	At home.
Dec. 1	70	100	60	1463	110.6	44.5	0	.....	24	At home.
1924.										
Jan. 1	70	100	60	1463	110.6	45.9	0	.....	24	At home.
Feb. 1	70	100	60	1463	110.6	47.3	0	.....	24	At home.

height, but the insulin dosage has remained exactly the same at 24 units per day with the same diet. The basal metabolism is now normal.

Case II.—Case No. 328, female, aged eighteen years. Admitted March 16, 1922. In January, 1921 the first symptoms appeared, she having had irregular treatment up until the time of admission. Physical examination was normal. Wassermann was negative. Chart II shows the subsequent course.

At the time of the first discharge (June, 1922) the diet contained only 15 grams of carbohydrate. Subsequently, even that small intake had to be decreased to maintain freedom from glycosuria. After readmission she was established upon a diet of 1567 calories with 20 to 24 units of insulin per day. Since discharge one year ago the insulin dosage has remained the same at 20 units per day.

A review of these 2 cases (I and II), both severe in young girls, both types that had given themselves every possible chance to improve by carefully regulated, weighed intakes,

CHART II

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C. H. O., gm.	Total calories.	Total glucose, gm.					
1922.										
Mar. 17	66	69	44	1093	89.18	38.7	70.5	0.34	0	Admission.
June 5	42	100	15	1164	49.36	36.3	0	0.13	0	Discharged.
Aug. 1	42	100	12	1151	46.36	39.7	0	....	0	At home.
Oct. 1	42	100	2	1110	36.36	40.0	0	....	0	At home.
Dec. 1	42	50	0	637	29.36	38.0	0	....	0	At home.
1923.										
Feb. 9	40	100	25	1197	58.2	36.1	26.7	0.34	0	Readmission.
Feb. 14	60	120	50	1567	96.8	36.5	0	0.19	40	
Feb. 23	60	120	50	1567	96.9	37.2	0	0.14	20	
Mar. 12	60	120	50	1567	96.8	38.2	0	0.14	24	
Mar. 20	60	120	50	1567	96.8	38.7	0	0.13	20	Discharged.
May 1	60	120	50	1567	96.8	42.7	0	....	20	At home.
July 1	60	120	50	1567	96.8	42.8	0	....	20	At home.
Sept. 1	60	120	50	1567	96.8	45.4	0	....	20	At home.
Nov. 1	60	120	50	1567	96.8	45.4	0	....	20	At home.
1924.										
Jan. 1	60	120	50	1567	96.8	45.9	0	....	20	At home.
Feb. 1	60	120	50	1567	96.8	46.0	0	....	20	At home.

extending over a period of years, shows that after establishment upon a suitable diet with necessary insulin dosage there has been practically no change in the individual's own pancreatic manufacture of insulin. No other conclusions can be drawn.

The second group of cases that I wish to show are ones in middle-aged individuals, not of the grade of severity as Cases I and II, all of which have shown definite improvement in their carbohydrate tolerance referable to insulin treatment.

Case III.—Case No. 169, female, aged fifty-three. Admitted November 18, 1921, with onset of symptoms in August, 1920. Glycosuria was found on October 20, 1920. Physical examination was unimportant. Wassermann was negative. Chart III (p. 1764) shows the course of the case.

After the first hospital period the diet was built up to 1843 calories without glycosuria, but subsequently it was impossible for her to keep sugar free with a considerably lower carbohydrate intake. Loss of weight began. With readmission she was established upon a diet of 2278 calories, requiring by discharge 17 units of insulin per day.

CHART III

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C. H. O., gm.	Total calories.	Total glucose, gm.					
1921.										
Mar. 19	85	60	57	1140	112.3	56.6	28.5	0.26	0	Admission.
Apr. 16	80	80	70	1359	124.4	54.0	0	0.13	0	Discharged.
Oct. 1	90	100	100	1709	163.2	56.4	0	....	0	At home.
1922.										
Apr. 1	90	110	110	1843	173.2	59.1	0	....	0	At home.
Oct. 1	90	110	110	1843	173.2	59.0	0	....	0	At home.
1923.										
Apr. 1	90	110	90	1761	153.2	59.5	0	....	0	At home.
Aug. 8	80	110	80	1679	137.4	51.8	13.3	0.25	0	Readmission.
Aug. 24	70	170	100	2278	157.6	54.4	0	0.14	36	
Aug. 30	70	170	100	2278	157.6	54.4	0	0.14	23	
Sept. 4	70	170	100	2278	157.6	55.1	0	0.17	17	Discharged.
Oct. 1	70	170	100	2278	157.6	57.2	0	....	15	At home.
Nov. 1	70	170	100	2278	157.6	56.8	0	....	10	At home.
Dec. 1	70	170	100	2278	157.6	60.0	0	....	10	At home.
1924.										
Jan. 1	70	170	100	2278	157.6	60.0	0	....	10	At home.
Feb. 1	70	170	100	2278	157.6	60.4	0	....	10	At home.

At home with the same diet 10 units have now kept the urine free from sugar for four months, and her weight has been built up to a normal level.

Case IV.—Case No. 330, male, aged thirty-three. First admitted to the hospital March 2, 1922, the history of diabetes dating from November, 1921. There had been marked loss of weight. Physical examination was unimportant and the Wassermann was negative. Chart IV follows:

CHART IV

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C. H. O., gm.	Total calories.	Total glucose, gm.					
1922.										
Mar. 22	87	92	58	1450	117.7	56.6	44.3	0.29	0	Admission.
Apr. 11	60	100	40	1340	84.8	54.7	0	0.11	0	Discharged.
Dec. 1	60	150	50	1846	99.8	56.0	0	....	0	At home.
1923.										
Mar. 1	60	150	50	1846	99.8	54.0	0	....	0	At home.
Nov. 16	58	126	58	1647	104.2	57.2	92.4	0.24	0	Re-admission.
Dec. 1	60	190	70	2300	123.8	61.1	0	0.16	55	Discharged.
1924.										
Jan. 1	60	190	70	2300	123.8	65.0	0	....	39	At home.
Feb. 1	60	190	70	2300	123.8	70.0	0	....	30	At home.

Case IV shows that the diet was built up to 1340 calories by the time of the first discharge. Living upon a weighed diet at home things went well until the summer of 1923, by which time the diet had been increased to 1864 calories. Then glycosuria returned and continued with marked dietetic restriction. Upon readmission November 16th there was marked glycosuria, 92.4 grams, upon a basal diet of 1647 calories. This was controlled by insulin, the diet being subsequently increased to 2300 calories, with a total glucose yield of 123.8 grams. Fifty-five units of insulin per day were required. Since discharge the diet has been kept the same, there has been a gain of 12.8 kilos in weight, and the daily insulin requirement has decreased to 30 units per day.

Case V.—Case No. 540, male, aged forty-six. Admitted May 18, 1923, the onset of symptoms—thirst and polyuria—being in September, 1921, when glycosuria was found. During the next eighteen months treatment was irregular. Loss of weight and polyphagia were marked. Physical examination showed a thin man, with marked secondary anemia. Blood showed lipemia. Wassermann was negative. Chart V shows the remarkable course of the case.

CHART V

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C. H. O., gm.	Total calories.	Total glucose, gm.					
1923.										
May 20	75	70	50	1164	100.5	50.1	76.7	0.28	0	Admission
June 3	85	170	60	2176	126.3	51.1	0	0.15	45	
June 12	90	180	75	2350	145.2	52.0	0	0.13	48	Discharged
July 1	90	180	75	2350	145.2	54.0	0	....	45	At home.
Aug. 1	90	180	75	2350	145.2	59.0	0	0.12	30	At home.
Sept. 1	90	180	75	2350	145.2	60.0	0	....	25	At home.
Oct. 1	90	180	75	2350	145.2	59.0	0	....	20	At home.
Nov. 1	90	180	75	2350	145.2	60.0	0	....	20	At home.
Dec. 1	90	180	75	2350	145.2	60.0	0	0.13	15	At home.
1924.										
Jan. 1	90	150	75	2072	142.2	61.0	0	....	0	At home.
Feb. 1	90	150	75	2072	142.2	60.0	0	....	0	At home.

By discharge, on June 12th, the patient was taking 2350 calories, about 1100 over his basal requirement figured on his

reduced weight. He was gaining weight and holding a normal blood-sugar with 48 units of insulin per day. Since discharge he has held the same diet except for a decrease since January 1st, maintained a normal weight, with normal blood-sugars upon decreasing insulin dosage. Since January 1st no insulin has been necessary.

Case VI.—Case No. 667, male, aged thirty. Patient was admitted to the hospital December 3, 1923, the diabetic symptoms only dating back to October 1, 1923. Physical examination revealed considerable loss of weight. The Wassermann was negative.

The essential data is presented in Chart VI.

CHART VI

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C. H. O., gm.	Total calories.	Total glucose, gm.					
1923.										
Dec. 4	54	118	54	1540	97.1	53.1	69.8	0.29	0	Admission.
Dec. 23	55	190	60	2239	110.9	55.0	0	0.20	46	
Dec. 28	65	190	65	2300	121.7	54.5	0	0.20	44	Discharged.
1924.										
Jan. 1	65	190	65	2300	121.7	57.3	0	....	40	At home.
Feb. 1	65	190	65	2300	121.7	57.7	0	....	20	At home.
Mar. 1	65	190	65	2300	121.7	58.0	0	....	18	At home.

Upon admission with a basal diet of 1540 calories there were 69.8 grams of glycosuria. By December 28th the diet contained 2300 calories, with a total glucose yield of 121.7 grams, it taking 44 units of insulin per day. Since discharge with the same intake the insulin dosage has been decreased rapidly to 18 units per day, with complete freedom from glycosuria.

A critical review of this last group of cases (III, IV, V, and VI) shows definite evidence of improvement in carbohydrate tolerance. This is of more significance in the first 3 cases, they being of longer standing, and having lived upon a period of weighed diet without insulin. Case V is most unusual, the

increase in tolerance being equivalent to at least 45 grams of carbohydrate (probably 72 grams) in a space of six months.

This aspect of insulin treatment offers great encouragement to many severe cases of diabetes mellitus. If the patient can anticipate reduction or stoppage of insulin dosage his outlook is much healthier.

Another question relative to insulin treatment that I wish to mention is that of the relation of weight to carbohydrate tolerance. How much weight shall we allow our patients to gain, and what will happen if they become too fat? Apparently it is a relatively easy matter to re-establish the nutrition of an underweight diabetic with insulin, but will overnutrition cause lowering of the carbohydrate tolerance of the individual? Dr. Joslin, as you know, has shown that individuals overweight are much more liable to become diabetic than if underweight, while the basis of Dr. Allen's therapy has always been undernutrition.

An example of the deleterious effect of overweight is well shown in the following case:

Case VII.—Case No. 502, male, aged nineteen. First admitted on November 27, 1922, with diabetes since November, 1919. Its course had been irregular, with glycosuria at intervals up until the time of admission. Physical examination showed marked loss of weight, and an extensive fibroid tubercular lesion was found in the upper lobe of the right lung. There was slight evening temperature, but sputum was negative to T. B. C. Wassermann was negative. The course is shown in Chart VII (p. 1768).

After the first discharge his course was downhill, with inability to remain free from glycosuria. Readmitted and established upon a diet of 1660 calories, with total glucose yield of 97.8 grams, the insulin dosage required was 34 units per day. With his gain in weight to date of 21.2 kilos the insulin dosage required has had to be increased to 55 units to maintain freedom from glycosuria. Further, the carbohydrate in the diet has been cut to 45 grams. It is interesting to note that with his

CHART VII

Date.	Intake.					Weight, kilos.	Glycosuria, gm.	Blood-sugar fasting.	Insulin units per day.	Remarks.
	Protein, gm.	Fat, gm.	C, H. O., gm.	Total calories.	Total glucose, gm.					
1922.										
Nov. 28	57	59	37	934	75.96	37.5	8.4	0.25	0	Admission.
Dec. 22	50	33	75	819	107.3	31.4	0	0.09	0	Discharged.
1923.										
Mar. 15	50	60	25	866	60.0	36.5	30.0	0.26	0	Readmission.
Apr. 1	60	130	50	1660	97.8	36.5	0	0.19	34	
Apr. 12	60	130	50	1660	97.8	37.4	0	0.12	34	Discharged.
Sept. 10	60	130	50	1660	97.8	52.0	0	0.25	46	At home.
Oct. 22	60	130	50	1660	97.8	55.2	36.5	0.25	45	At home.
Nov. 5	60	130	50	1660	97.8	56.0	0	0.20	45	At home.
Dec. 1	60	130	55	1681	102.8	56.5	+	....	50	At home.
1924.										
Jan. 1	60	130	45	1640	92.8	56.5	+0	....	55	At home.
Feb. 1	60	130	45	1640	92.8	57.7	+0	....	55	At home.

improved state of nutrition all activity of the pulmonary lesion has disappeared, but *x*-rays show the same findings. The patient today is overfat.

Thus evidence has been presented which indicates that insulin *per se* will in some severe cases of diabetes mellitus cause an increase in the carbohydrate tolerance, also it is unwise to allow patients to gain more than their theoretic weight, the excess acting as an increased burden upon their pancreatic function, resulting in actual lowering of their carbohydrate tolerance.

CLINIC OF DR. H. P. WRIGHT

ROYAL VICTORIA HOSPITAL

THE EFFECT OF LIGHT IN THE TREATMENT OF DISEASE[1]

THE subject for consideration today is too vast for proper presentation in the amount of time at my disposal, and therefore, unquestionably, many diseases in which light has a very distinct therapeutic effect will not be discussed.

In dealing with a therapeutic agent such as light, which has for so many years been prostitutized by the quacks and "hangers-on" of our profession, it behooves us all to adopt a conservative attitude; but, we are not justified in the present, because of the past, in shutting our eyes to the opening of a new chapter in the treatment of certain diseases. That in the actinic ray we have a panacea for all the ills of the flesh would be a ludicrous and malignant untruth. Nevertheless, the fact remains that a great many diseases are benefited by judicious ultraviolet therapy.

Case I.—The following is a good example of the effect of ultraviolet therapy from the mercury vapor lamp in multiple tuberculosis.

No. 39,279, A. G., three-and-a-half years of age, was admitted to the service of Dr. S. G. Ross in the pediatric Ward of the Royal Victoria Hospital on November 22, 1923. The child is said to have been well until January, 1922, when he suffered from an attack of measles. About six weeks later he developed abscesses on the right elbow and on the face close to the angle of the mouth, and some weeks later a tuberculous

[1] From the Pediatric Clinic of the Royal Victoria Hospital, Montreal.

lesion in the left foot. Previous to admission to the Royal Victoria Hospital he spent some months in other hospitals.

The *physical examination* on admission showed an emaciated child with tuberculous ulcers distributed in the following manner: One over the right malar bone, two over the right shoulder, one over the right elbow, one over the right knee and right ankle, also over the left elbow, left knee, and left ankle. Some were dry and covered with scabs, while others were discharging and ulcerating.

The *lymphatic system* showed generalized enlargement of the superficial glands approximating the various skin and bone tubercular lesions.

On examination of the *lungs* sibilant and fine râles were heard over the right side posteriorly and under the right axilla.

The *abdomen* was tender to deep palpation; there was no rigidity and no masses were felt. There were no signs of free fluid. The lower edge of the liver and spleen were palpable. The other system showed no abnormal signs.

Urine negative.

Blood (November 23, 1923): Erythrocytes, 4,460,000; leukocytes, 18,400; hemolgobin, 70 per cent.; Wassermann negative.

Diary (November 24th): Positive intradermal reaction to O. T. 1/10 milligram.

First exposure to ultraviolet ray (mercury vapor lamp):

December 6, 1923 chest clearing.

December 12, 1923 Sir Henry Gray in consultation confirmed the diagnosis, and the following is an extract from a note made by him on December 18, 1923:

"Diagnosis, Multiple tuberculosis.

"Foci: The tarsal bones of both feet were found to be in a state of pulp; the bones adjacent to the right elbow, ditto; sinuses leading down to these three bony areas cureted and a considerable amount of bony débris removed from each. Packed with iodoform gauze in each case. The proximal phalanx of the right middle finger was so disintegrated that the finger was removed at the metacarpal phalangeal joint. Iodoform gauze

pack; flaps not sutured. Numerous other sinuses and ulcerated areas, some leading down to abscess in the depth, were rapidly scraped, and iodoform gauze packed in. Both lower extremities and the right upper extremity were cast in plaster. It is hoped to be able to leave the child without dressing the wounds

Fig. 294.—Case I. Photographed March 6th. Note second fingers on both hands and general appearance of child.

for at least a week. The patient became rather collapsed during operation, so that the cureting had to be done very rapidly.

"January 8, 1924: Condition of the child and wounds greatly improved. Strips of gauze dipped in B. I. P. reintroduced, but in smaller amount. Stump of amputated finger soundly healed."

On this occasion it was felt that the plaster of Paris was preventing radiation of the extremities, and on that account

modified and simpler splints were applied to enable a greater utilization of ultraviolet therapy.

The skiagrams and pathologic reports confirmed the diagnosis of tuberculosis, and the *x*-ray plates were interpreted as

Fig. 295.—Case I. Photographed March 6th. Showing healed ulcers.

showing active disease of the bone in several localities. Unfortunately space will not permit of showing the improvement as demonstrated by *x*-ray plates.

Temperature Chart.—During the first six weeks the temperature varied between 99° and 104.4° F. and for several days

between 101° and 104° F. During the last four weeks it has seldom been above 100° F., and has, as a rule, fairly closely followed the normal line. The table on pages 1774 and 1775 gives in detail the ultraviolet therapy.

The photographs (Figs. 294–296) show the child as he is today, with all superficial lesions healed.

Skiagraphs of the lungs on various occasions were negative for active disease, and physical examination of the lungs at present reveals no adventitious sounds. It would appear as

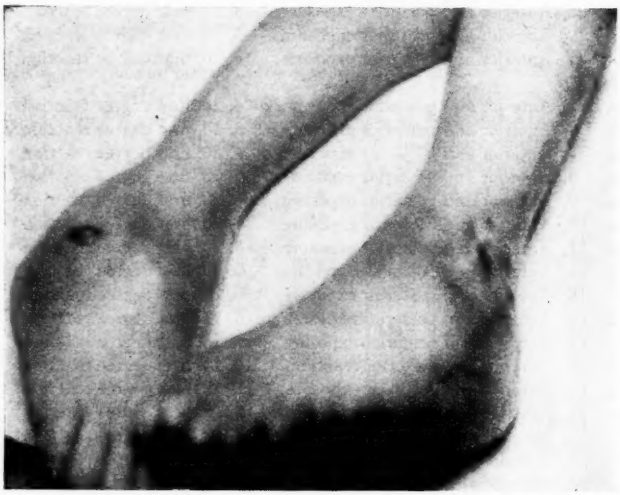


Fig. 296.—Case I. Photographed March 6th. Showing healed ulcers on feet.

though the sounds found on admission were probably caused by some acute respiratory infection.

Discussion.—The above case is a good example of the effect of ultraviolet therapy in multiple tuberculosis, and I saw many such cases last summer at Rollier's Clinic in Leysin.

It would be unwise to assume that ultraviolet therapy without adjuncts would have been successful in this case, for a very necessary adjunct was provided in that the patient was supplied with an abundance of *fresh air*.

A. G., ULTRAVIOLET RAY TREATMENT FROM MERCURY VAPOR LAMP

		Minutes.	Distance, inches.	
1923.				
Nov. 24,	Anterior and posterior exposure,	āā 5,	25,	second degree reaction
Nov. 27,	Partial exposure,	5,	20,	first degree reaction
Nov. 29,	Anterior and posterior exposure to feet,	āā 5,	20,	first degree reaction
Dec. 17,	Anterior and posterior exposure (without feet),	āā 1,	20,	first degree reaction
Dec. 20,	Anterior and posterior exposure,	āā 2,	20,	first degree reaction
Dec. 22,	Anterior and posterior exposure,	3,	20,	first degree reaction
Dec. 26,	Anterior and posterior exposure,	3,	20,	first degree reaction
Dec. 29,	Anterior and posterior exposure,	3,	20,	first degree reaction
Dec. 31,	Anterior and posterior exposure,	4,	20,	first degree reaction
1924.				
Jan. 2,	Anterior and posterior exposure,	āā 4,	20,	first degree reaction
Jan. 4,	Anterior and posterior exposure,	3,	20,	first degree reaction
Jan. 7,	Anterior and posterior exposure,	4,	20,	first degree reaction
Jan. 8,	Anterior and posterior exposure,	5,	20,	first degree reaction
Jan. 9,	Anterior and posterior exposure,	6,	20,	first degree reaction
Jan. 10,	Anterior and posterior exposure,	7,	20,	first degree reaction
Jan. 11,	Anterior and posterior exposure,	8,	20,	first degree reaction
Jan. 14,	Anterior and posterior exposure,	9,	20,	first degree reaction
Jan. 15,	Anterior and posterior exposure,	6,	18,	first degree reaction
Jan. 16,	Anterior and posterior exposure,	8,	18,	first degree reaction
Jan. 17,	Anterior and posterior exposure,	9,	18,	first degree reaction
Jan. 18,	Anterior and posterior reaction,	10,	18,	first degree reaction
Jan. 19,	Anterior and posterior exposure,	11,	18,	first degree reaction
Jan. 21,	Anterior and posterior exposure,	12,	18,	first degree reaction
Jan. 22,	Anterior and posterior exposure,	13,	18,	first degree reaction
Jan. 23,	Anterior and posterior exposure,	14,	18,	first degree reaction
Jan. 24,	Anterior and posterior exposure,	15,	18,	first degree reaction
Jan. 26,	Anterior and posterior exposure,	15,	18,	first degree reaction
Jan. 28,	Anterior and posterior exposure,	15,	18,	first degree reaction
Jan. 29,	Anterior and posterior exposure,	15,	18,	first degree reaction
Jan. 30,	Anterior and posterior exposure,	15,	18,	first degree reaction
Jan. 31,	Anterior and posterior reaction,	15,	18,	first degree reaction
Feb. 1,	Anterior and posterior exposure,	15,	18,	first degree reaction
Feb. 2,	Anterior and posterior exposure,	15,	18,	first degree reaction
Feb. 4,	Anterior and posterior exposure,	15,	18,	first degree reaction
Feb. 5,	Anterior and posterior exposure,	10,	18,	first degree reaction
Feb. 6,	Anterior and posterior exposure,	10,	18,	first degree reaction
Feb. 7,	Anterior and posterior exposure,	10,	18,	first degree reaction
Feb. 8,	Anterior and posterior exposure,	10,	18,	first degree reaction
Feb. 9,	Anterior and posterior exposure,	10,	18,	first degree reaction
Feb. 11,	Anterior and posterior exposure,	10,	18,	first degree reaction

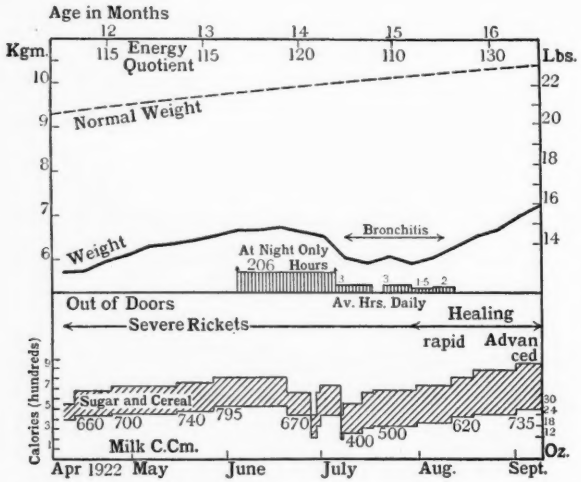
1924.		Minutes. Distance, inches.
Feb.	12, Anterior and posterior exposure, āā	10, 18, first degree reaction
Feb.	13, Anterior and posterior exposure,	10, 18, first degree reaction
Fdb.	14, Anterior and posterior exposure,	10, 18, first degree reaction
Feb.	15, Anterior and posterior exposure,	10, 18, first degree reaction
Feb.	16, Anterior and posterior exposure,	10, 18, first degree reaction
Feb.	18,[1]Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	1, 18, first degree reaction
Feb.	19, Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	2, 18, first degree reaction
Feb.	20, Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	3, 18, first degree reaction
Feb.	21, Anterior and posterior reaction,	10, 18, first degree reaction
	Arm and legs exposure,	4, 18, first degree reaction
Feb.	22, Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	5, 18, first degree reaction
Feb.	23, Anterior and posterior exposure,	10, 18, first ddgree reaction
	Arm and legs exposure,	6, 18, first degree reaction
Feb.	25, Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	7, 18, first degree reaction
Feb.	26, Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	8, 18, first degree reaction
Feb.	27, Anterior and posterior exposure,	10, 18, first degree reaction
	Arm and legs exposure,	9, 18, first degree reaction
Feb.	29, Anterior and posterior reaction,	10, 18, first degree reaction
	Arm and legs exposure,	10, 18, first degree reaction
Mar.	1, Anterior and posterior exposure,	10, 18, first degree reaction
Mar.	2, Anterior and posterior exposure,	10, 18, first degree reaction
Mar.	4, Anterior and posterior exposure,	10, 18, first degree reaction
Mar.	5, Anterior and posterior exposure,	10, 18, first degree reaction
Mar.	6, Anterior and posterior exposure,	10, 18, first degree reaction

The dosage of ray therapy had to be carefully controlled, and on several occasions there was a definite rise in temperature following treatment.

Finally, although the case has been so successfully treated, pigmentation of the skin has never been pronounced. Generally speaking, one may say that radiation from the sun and carbon-arc lamp cause much more marked pigmentation than radiation from the mercury vapor lamp.

[1] Right arm and both legs put in plaster December 18th until February 18th.

Case II.—The following case is taken from Special Report Series No. 77, Medical Research Council. Studies on Rickets in Vienna, 1919–22.



A. W. Case II

Admitted with rickets. The bone lesions were not improved by outdoor treatment at night in summer, but healed rapidly when the child was exposed to sunshine.

Born. 24th April *1921*.

8th April *1922*. Admitted, aged 11 months. Received diet of full milk and carbohydrate (see note below). Child small for age and undernourished. Craniotabes, rosary, and enlarged epiphyses present. X-ray plates show advanced rachitic changes in epiphyses.

3rd June. No improvement. Child's cot placed out of doors at night.

6th July. X-ray plate shows no evidence of healing after 5 weeks of outdoor treatment at night. Has been out on 25 nights for a total of 206 hours. Outdoor treatment by day begun with exposure to sunshine.

29th July. Has had acute bronchitis. Radiographic evidence of healing of bone lesions after 70 hours out of doors. (On account of bad weather, out of doors only on 14 out of 23 days.)

12th August. Unusually rapid healing of bone lesions.

17th September. Age 17 months. Healing of bone lesions far advanced. Can sit alone; general condition good.

Note. For eleven weeks after admission the milk given to this child was obtained from stall-fed cows receiving green fodder daily. No improvement in bone lesions took place on this diet.

Fig. 297.

Discussion.—In the Vienna experiment quite as satisfactory results were obtained by the employment of the mercury vapor lamp, and I have only selected the above case in order to demonstrate treatment by heliotherapy as well as by artificial ultra-violet therapy.

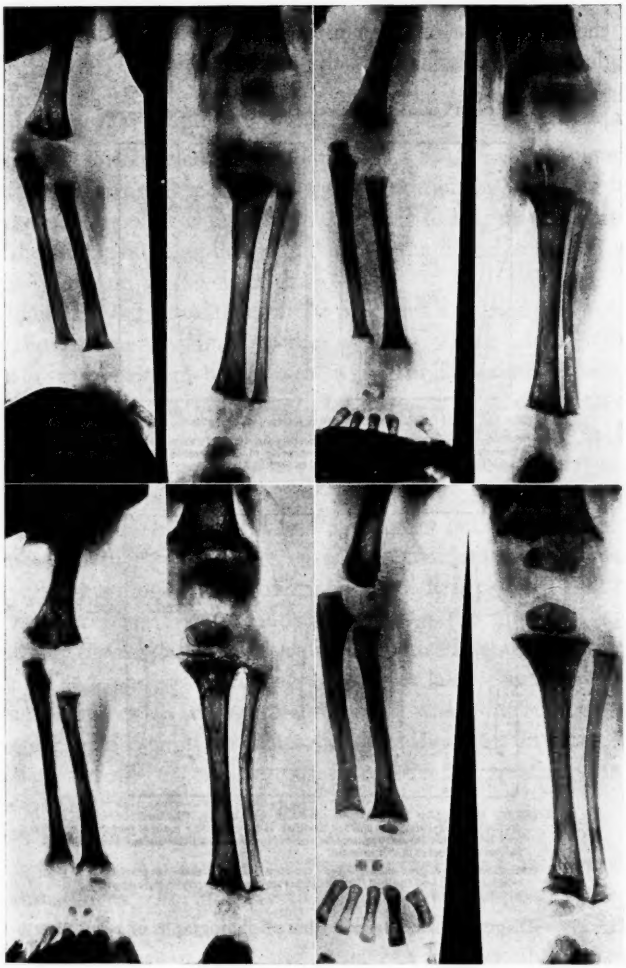
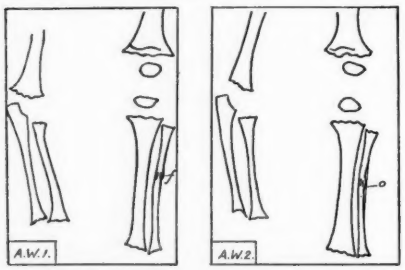


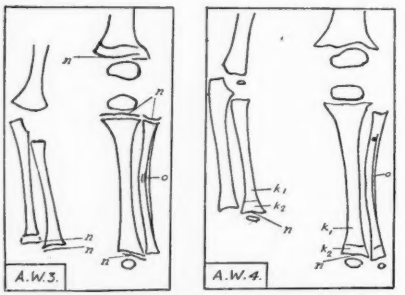
Fig. 298.—A. W. Case II. Admitted with rickets and treated out-of-doors with exposure to sunshine.

It is not quite clear to whom we are responsible for the pioneer work in clinical heliotherapy. Dr. O. Bernard, of St. Moritz (Switzerland), claims that his observations date from 1902, and

Rollier claims that in 1903 he opened at Leysin the first clinic for the application of the sun-treatment, and it is he who has taught us to use sunlight wisely, but intensively.



1. (22.4.22.) Florid rickets with a greenstick fracture (*f*) of the fibula.
2. (29.6.22.) No alteration in condition except possibly the formation of a trace of periosteal osteoid tissue (o) in the concavity of the fractured fibula after 2½ months in hospital in spite of outdoor treatment by night.



3. (5.8.22.) Healing after 30 days' treatment out of doors by day. The band of newly calcified tissue (o) along the shaft of the fibula is distinct. New zones of preparatory calcification (*n*) are apparent at the ends of almost all the shafts. Rarefaction of the bones is still extreme.
4. (17.9.22.) Good progress shown. Note the clearly defined zones of provisional calcification (*n*) and the finely meshed structure of the metaphyses (k_2) compared with the diaphyses (k_1).

Fig. 299.—Diagrammatic interpretation of photographs of *x*-ray plates.

Last summer I had an opportunity of visiting his clinic, and I am filled with admiration for the great work that he is accomplishing at Leysin. Rollier was unquestionably the first to advocate the general sun bath as distinct from the local application of sunlight.

From the purely scientific side it is interesting to recall that Charcot, in 1859, showed that the effect of certain light rays on the skin is independent of any heating action. Downes and Blunt published their researches on the bactericidal properties of light in 1877, and in 1893 Finsen's first series of experiments appeared, forming the main scientific basis of ultraviolet therapy.

A momentary inspection of the diagram of the spectrum will make it clear with which rays we appear to be most concerned in the therapeutic application of light—the invisible rays in the ultraviolet end of the spectrum.

These rays, fortunately, can be produced artificially by means of the carbon-arc lamp or the mercury vapor lamp. Glass which may intervene between the source of light and the patient deflects most important rays. In the case of the carbon-arc lamp this feature is of no particular importance; but in the mercury vapor lamp quartz has to substitute glass as a receptacle for the mercury.

At the Finsen Institute in Copenhagen light from the carbon-arc is most successfully used for the treatment of skin, bone, and lymph tuberculosis, and there it is claimed that the carbon-arc lamp is more satisfactory than the mercury vapor lamp for the reason that it is said to produce a more penetrating ray. The exponents of the mercury vapor lamp claim that their lamp is richer in ultraviolet radiation than the carbon-arc. However, no definite evidence has been brought forward, and to date, for all practical purposes, the effect of treatment from the two lamps seems to be identical.

All artificial methods for producing ultraviolet rays are an attempt to simulate solar rays under the sun's most favorable circumstances. Fortunately the sun is at the disposal of us all for a part of the year, and during the season that it is impossible to utilize heliotherapy many of us can employ the mercury vapor or the carbon-arc lamp. The mercury vapor lamp is now being produced by several firms, and the purchase cost in the United States varies from about $330 to $500. It is simple to operate.

Certain conclusions have been arrived at in heliotherapy

from meteorologic measurements which have been summarized by Rosselet in the following manner:

I. Direct light:

A. *At a given place (plain or high altitude):*

(*a*) The intensity of the different portions of the solar spectrum is approximately the same in summer.

(*b*) The differences between these partial intensities increase as winter is approached. The shorter the wave-length under consideration, the more marked is this seasonal variation. The winter sun is poor in ultraviolet rays.

B. *The difference between high and low altitudes:*

(*a*) *In summer* the intensity of the different radiations is approximately the same at high and low altitudes.

(*b*) *In winter* the value of this intensity diminishes, but at high altitudes to a less extent than in low country. This comparatively small degree of seasonal difference in the intensity of the solar radiations is an important characteristic of the climate of high altitudes. As a result of this fact, the intensity of solar radiation is more equable throughout the year in high altitudes than in low country, and heliotherapy may be practised there in all months of the year.

These facts show that heliotherapy, considered solely from the point of view of solar radiations, may in summer be practised in low country just as well as at high altitudes. In this season the advantages of the mountain air are to be looked for in the cool, dry, bracing quality of its air, comparing favorably with that of the plain, which is apt to be warm and moist and to have an enervating action which may counteract the good effects of the treatment.

Sea air has, in summer, great advantages over inland air.

Physiology.—How light produces its therapeutic effect has not yet been determined; but this problem is engaging the attention of many investigators all over the world, and we may confidently expect some results before many years. Downes

and Blunt[1] in 1877 proved the bactericidal power of light by the following experiment: Culture-tubes were divided into two lots, the first consisting only of plain tubes, while in the second the tubes were surrounded by a sheet of lead foil which would prevent the action of light without interfering with that of heat. When exposed to light for a given time it was found that only in the tubes surrounded by lead had the germs developed. It was also shown that light had not modified the culture-medium in the tubes which had remained sterile, as subsequent inoculation of these resulted in abundant cultures.

Since then numerous other workers have investigated this problem, and today opinions are united in recognizing the powerful bactericidal power of the ultraviolet group of radiations.

The question of penetration of ultraviolet rays is still undecided. Leonard Hill[2] in 1920 demonstrated the repeated observation of the radiations of short wave-lengths by the integument, and that the possibility of deep penetration must be looked for among the radiations of greater wave-lengths.

Although the direct action of light is often disputed, this is not the case with the indirect action, which is universally accepted. In 1914 Guie[3] reported the case of a child suffering from tuberculosis of the tracheobronchial glands who tolerated moderate sun treatment very well on feet and legs. The mother, on her own initiative, doubled the duration of insolation. The change was followed by a severe cough, rise in temperature, and all other symptoms which characterize the reaction of a glandular lesion in this locality.

The rôle of pigmentation is still uncertain. Its appearance coincides with the action of substances which irritate the skin. Certain writers have recorded pigmentation as harmful in ultraviolet therapy on account of interference with the penetration of radiations. Clinically, this does not seem to be borne out, as pigmentation is almost a therapeutic index of cure. Possibly pigmentation may materially help by protecting the body from too great heat; or again, pigmentation may act in a similar manner to fluorescent substances.

Exactly what rôle fluorescent substances play in light therapy has not yet been determined. It is possible that they may be of extreme importance and capable of transforming rays of short wave-length into longer and more penetrating ones. If this proves to be the case it will be a fact of first-rate clinical importance, for it may enable us to employ solar rays after they have traveled through glass. *In simple language this means that we would be enabled to employ heliotherapy throughout the year in Canada or the United States.*

The action of light on blood-cells has been the subject of many investigations. Unfortunately the evidence is conflicting and no definite conclusions can be made. In many cases blood changes may be due to the beneficial action of light therapy on the general health rather than to any action of the ultraviolet rays on the hematopoietic system.

Treatment by Light.—From the foregoing remarks it will be readily understood that the technic of applying light is extremely important. One is too ready to assume that light therapy is a simple, common-sense matter which any fool can apply. This is very far from being the case, and in all probability many have lost faith in ultraviolet therapy on account of failure to appreciate this point. All cases treated at Leysin by heliotherapy are kept under observation indoors for at least forty-eight hours before receiving insolation, and on the first day of treatment they are allowed only five minutes' exposure to the solar rays. The insolation is gradually increased until three hours daily is being received. It has been found that more than three hours' insolation is unnecessary, and even in some cases harmful.

The following extract from Rollier's[4] text-book gives a good idea of how to employ heliotherapy.

"**Technic of the Sun-bath.**—The technic we have finally adopted for the sun-bath has been evolved during a number of years of experience, and is the one which we have found empirically to be the safest; this method consists in beginning always with the feet and insolating both legs and arms before exposing the abdomen and thorax. By this means we are able

to get a general idea of the tolerance of the patient to sunlight before the more vulnerable parts of the body are exposed; any accidents which result are, therefore, of a slight nature, and as they only affect outlying regions of the body, their general effects are likely to be minimal. The thoracic and abdominal viscera are not subjected to any congestion, but rather to a decongestive action, as insolation of the extremities causes the blood to flow to these regions and therefore away from the viscera.

"We must also emphasize the importance of using short periods of insolation (ten to fifteen minutes), alternating with periods of rest (five to ten minutes). These short periods of rest enable the body to tolerate a much greater total amount of sunlight than would be possible with one long sun-bath, which is much more fatiguing to the patient, besides being more irritating to the skin. We find that a series of moderate reactions three or four times a day (ten to fifteen minutes) is more beneficial and causes more rapid acclimatization than is the case with a single period of thirty minutes. The vasomotor system of the skin covering those parts of the body usually under the clothes is much less developed than that of the exposed parts, and reaction to stimulation is comparatively lethargic; by exposing the skin of these regions several times a day this vasomotor reaction is called into play and with use takes place with greater facility. The skin, subcutaneous tissues, and muscles are much benefited by this improved circulation, and their development, especially that of the muscles, is very considerable. With patients suffering from visceral or pulmonary tuberculosis the necessity for this method of insolation is particularly great, as serious accidents may follow the congestion produced by faulty technic.

"The diagram (Fig. 300) shows the usual progression in a normal case; it does not represent a hard-and-fast rule, as we have here to take account of so many all-important and variable factors, such as general condition of the patient, localization and character of the lesion, and tolerance to sunlight, and besides these we must allow for various atmospheric conditions,

e. g., temperature and movement of the air, height of sun, and clearness of atmosphere (Figs. 300–303).

"Under the average conditions assumed in the diagram, the feet are uncovered on the first day three times for five minutes, on the second day three times for ten minutes, and on the third day three times for fifteen minutes, and so on. On the second day the legs are exposed at the same time as the feet, but only for five minutes; on the third day the thighs are similarly uncovered for five minutes, while the legs have ten minutes and the feet fifteen.

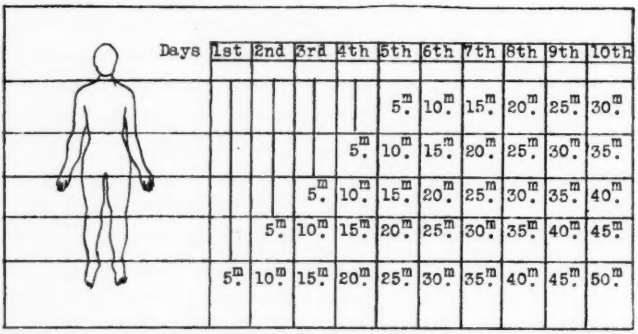


Fig. 300.—Diagram showing how progressive insolation should be carried out. From the tenth to the fifteenth day the same plan is followed. After the fifteenth day complete insolation from the beginning of the sun-bath. *Duration* three to four hours.

"On the fifth day, for example, the patient will have, at ten-minute intervals, three sun-baths of twenty-five minutes' duration, uncovering first the feet, and then, at five-minute intervals and in the following order, the legs, thighs, abdomen, and thorax. In cases complicated with pulmonary tuberculosis even slower progress is necessary. Where there is any cardiac disease or simply tachycardia a white cloth should be placed over the cardiac region. The head and nape of the neck should always be protected by a white linen hat, which should, if possible, be lined with green gauze, as this is more restful to the eyes than white. Smoked, dark yellow, or black glasses should also be used to protect the eyes.

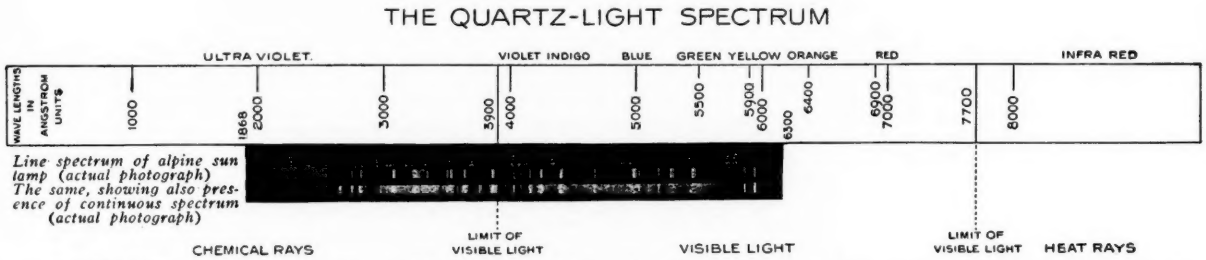


Fig. 301.—The above plate shows the visible and ultraviolet section of the electromagnet spectrum. The spectrum as shown includes part of the infra-red, the visible, and the ultraviolet rays. The colors of the spectrum are written above their respective wave lengths.

The photographic reproduction of the actual spectrum of the Alpine Sun Lamp gives a clear idea of the richness of the lamp in the ultraviolet region. It will be seen also from the lower photograph that a marked continuous spectrum is present in the lamp ray, as also in natural sunlight. It will be noticed that all that portion of the rays of the lamp falling to the left of the line, 3900 Angstrom units, cannot be detected at all by the human eye.

In order that it will be readily understood just what dimensions we are dealing with, it might be mentioned that the bright line shown at 3022 Angstrom units would correspond to approximately 175/1000 inch in length.

"If all goes well for ten days or so, the periods of insolation may be lengthened and reduced to two or three in number; by this time any idiosyncrasies of the patient will have been discovered and a fairly accurate estimate of his tolerance to sunlight formed; it will, therefore, be possible to hurry on the treatment with some patients while continuing cautiously with others. The duration of the sun-bath must also depend on the rate at which pigmentation takes place; where there is early pigmentation rapid advance may be made, while with patients

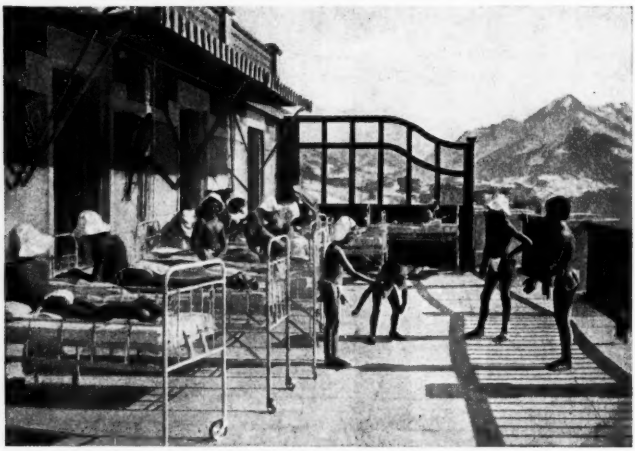


Fig. 302.—A solarium for children, Leysin, Switzerland.

whose skin reddens rather than browns there is danger of erythema, and caution is necessary. If for any reason the patient has had to interrupt treatment before he is properly pigmented, a start must be made several stages behind the previous maximum.

"When once the skin is well pigmented all over the body there is no longer any danger of overexposure, and the patient may have several hours of sun-cure every day. Three hours a day is for the majority of people the most suitable amount of exposure to sunlight; indications to exceed this only exist

in young persons whose bodies adapt themselves easily to their new surroundings and who pigment well. As a general rule sun-baths should be of shorter duration in summer than in winter, as in the former season the temperature of the air being itself comparatively high the additional heat of the sun is not so well tolerated."

All forms of tuberculosis except active pulmonary and meningeal are greatly improved by ultraviolet therapy. In active pulmonary tuberculosis and in tuberculous meningitis ultraviolet therapy is contraindicated. Bone tuberculosis, skin tubercu-

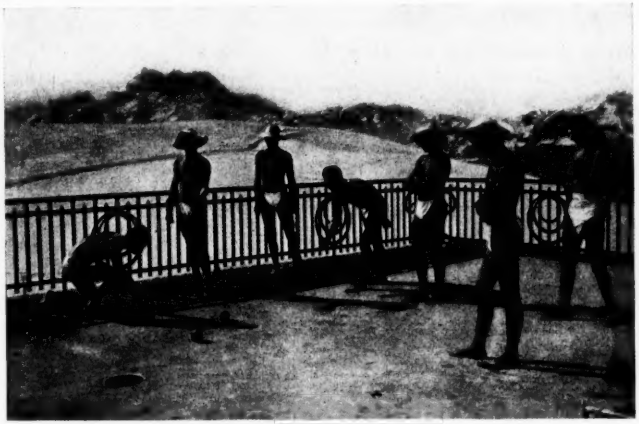


Fig. 303.—Convalescents playing games on the solarium roof of a clinic Leysin, Switzerland.

losis, tuberculous adenitis, tuberculous peritonitis, and pleurisy with effusion are among the forms most successfully treated.

Rickets is, perhaps, the most outstanding example of disease, other than tuberculosis, which is successfully treated by ultraviolet therapy.

Several skin diseases, such as acne vulgaris, psoriasis, pityriasis, and lupus, are strikingly improved.

We are justified, therefore, in concluding that ultraviolet therapy has a definite clinical application in the treatment of disease. In the absence of heliotherapy, ultraviolet radiations

from either the carbon-arc or the mercury vapor lamp is the logical substitute; but, with artificial ultraviolet therapy, *fresh air* must be an important adjunct of the treatment.

Finally, it is conceivable that as knowledge increases in connection with fluorescent substances it may be possible to reconvert rays of longer length, and so we may be enabled to use solar rays after their transmission through glass.

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CLINIC OF DR. FRED H. MACKAY

MONTREAL GENERAL HOSPITAL

PITUITARY DYSFUNCTION

THE several cases I wish to outline this afternoon will serve to illustrate some of the more important dysfunctions attributed to the pituitary gland.

This gland, as you already know, consists of two essential lobes, the anterior or pars glandularis and the posterior or pars nervosa, while partially enveloping the latter we have a thin cellular envelope called the pars intermedia. Developmentally, the anterior lobe, which may be regarded as the gland proper, takes its origin from the lining epithelium of the buccal cavity, while the posterior lobe is derived from and connected with the brain. This connection is affected through the infundibular stalk which permits of free communication with the ventricular cavities of the cerebrum. Situated within the sella turcica, an unyielding bony cavity which closely envelops this all-important organ, richly supplied with a sensitive sympathetic and blood mechanism, it is apparent that nature has excelled itself in its universal effort at protection.

And yet this very protective feature of the sellar cavity often spells disaster to what may be called normal function of the gland.

At certain physiologic periods in the life of the organism fluctuation in the size of the gland takes place.

During adolescence, menstruation, sexual indulgence, and after or accompanying periods of unusual mental excitement the gland becomes hyperemic and expands. If, too, during these periods one has an abnormally small sella to deal with, it is obvious that a general compression of the gland follows and

normal function is impeded. Should this impediment be inordinately prolonged, it is but a step from normal function to dysfunction—from a physiologic to a pathologic gland. Furthermore, this passage from physiologic to pathologic activity may entail the bursting forth of the irritated gland from the confines of its sheltered home. It may erode and destroy the walls with their terminal battlements—the clinoid processes—and either in the form of a simple hyperplasia or under the guise of adenoma, cystic degeneration, etc., may encroach upon and destroy such neighboring structures as the chiasm, the optic tracts, and some or all of that group of nerves and vessels enclosed within the cavernous sinus, namely, the third, fourth, sixth, and ophthalmic division of the fifth cranial nerves.

The destruction of these local structures, among which we may refer to pressure upon the pyramidal tracts from posteriorly directed growth, leads to a group of local disturbances which may be considered apart from the general endocrinous symptomology.

What, then, are the functions which may be interfered with, and to what part of the gland may their production be attributed?

The most important function of the anterior lobe is, no doubt, the control of growth, and some of the most spectacular expressions of perverted functions are seen in disturbance of this control. Growth and its control are not, however, the prerogative of the pituitary gland. While, doubtless, this gland is the chief arbiter in all growth processes, we must not forget that other glandular bodies, such as the adrenal cortex, the thyroid, and the interstitial cells of the testes, exert a very important influence in this direction.

Let us, at this stage, clearly understand that we cannot isolate the different endocrine glands into separate and individual secretory tight compartments.

Recent thought would tend to divide them into two antagonistic groups, the one under control of the sympathetic nervous system and comprising the pituitary, the adrenal, and the thyroid; the other governed by the autonomic system, which

has its influence with the pancreas and other digestive organs. Be this as it may, we are at the present time justified in assuming a state of physiologic reciprocity between the thyroid, adrenal, and pituitary glands.

Just how or to what extent this compensatory alliance may operate must yet be told, but so far it is clear that anomalies in growth may be set up by disturbed function in any one of these three glands. Furthermore, it is noted that a deficiency in anterior lobe secretion may be compensated for by oversecretion of the thyroid, at least in so far as it effects the development of skin, hair, and connective tissue. Likewise, the function of the posterior lobe in maintaining blood-pressure may, in case of need, be taken on to some extent by the chromaffin cells of the adrenal medulla.

And yet, after giving full credit to these allied endocrinous glands, we recognize in the secretion of the anterior lobe of the pituitary gland the predominant agent in the government of bodily development.

A second important function attributed to the anterior lobe is that of genital development. It stimulates the secondary sexual characteristics and, in cases of overactivity, accentuates the male markings.

A most important function is that of control of carbohydrate metabolism which is probably attributable to the pars intermedia, though many authorities regard this function as closely associated with the posterior lobe. We are, perhaps, on safer ground if we attribute these two last functions to the gland as a whole.

The posterior lobe is credited with the production of a secretion which stimulates the sympathetic nervous system and its subservient plain muscle tissue.

Thus, it stimulates uterine contraction—a function utilized so effectively by the obstetrician—it stimulates peristalsis—a fact not overlooked by the internist—and it increases arterial tension and plays, probably, the major rôle in the continued and regulated maintenance of blood-pressure—the reassuring ally of the surgeon.

Pathologic change in part or of the whole gland is obviously followed by disturbance in one or all of these secretory activities, and while, theoretically, we are apt to speak of hyper- or hypo-activity as if they were clean-cut clinical entities, practically every case shows the markings of both hyper- and hypofunction at some stage of its development.

Furthermore, the clinical expression of pituitary dysfunction differs entirely, according to whether it has its origin before or after puberty.

If then we bear in mind the fact that any one case may present a varied symptomatology, that signs of hyperfunction may coexist with those of hypofunction, that evidence of anterior lobe disturbance may complicate the typical picture of posterior lobe dysfunction, and finally, that the dividing line of puberty may alter the course and picture of the disease, we may with a degree of safety illustrate the various dysfunctions of the gland as a whole in diagrammatic form:

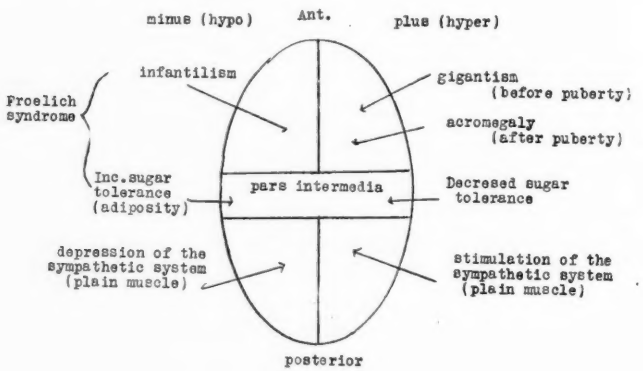

Fig. 304.

Case I.—Our first case is one which you will recognize at a glance.

The moderate frame, large head with protruding superciliary ridges, the prominent, square jaw, and the proportionately large hands stamp it as one of acromegaly.

He comes to us complaining of bitemporal headache and facial pain. Aged forty-two, he served three years in the Great War, during which time he was advanced to the rank of sergeant.

During the last year of his service in France he had great difficulty in procuring suitable boots. He noticed that almost every issue was necessarily larger. He now requires a No. $11\frac{1}{2}$ boot and a No. 10 glove, an increase of two sizes in the past four years.

The facial markings are pathognomonic. The eyes are deeply set under heavy protruding ridges. The glabella is thick and prominent, the jaw square, broad, and prominent—the prognathous type. The skin is coarse and deeply marked. The hair is abundant, coarse, and lavish in its distribution.

The external genitalia are of normal development, and yet he offers the information that his sexual power is far from what it used to be, a fact which he mistakingly attributes to his age. If we compare the length of the limbs with that of the trunk, they would not appear to be disproportionate, but we are impressed with the ponderousness of the distal parts. The head, hands, and feet appear to be suspended by altogether inadequate support.

x-Ray examination of the skull shows the bony markings as outlined and the sella turcica to be rather larger than the normal, with well-defined walls and intact clinoid processes.

Wassermann reaction is negative.

Urine is negative except for the finding of an intermittent glycosuria. The ingestion of 50 grams of glucose was followed by marked reduction of Fehling's solution and traces of sugar, over forty-eight hours.

Examination of the fields of vision does not show alteration from the normal, and no pallor of the disks can be made out.

Mentally, he is keen and alert, with a tendency to jocularity. His bearing is that of one who has been accustomed to take a leading place among his fellows, an observation which finds support in his overseas history.

This then is a typical picture of hyperactivity of the anterior

lobe, having its origin after puberty and after epiphyseal ossification has taken place—clinically acromegaly.

Case II.—This case is that of a young man twenty-five years of age and 5 feet, 8½ inches in height, who, too, served as a minor in the Great War. His complaints are headache, loss of sight in the left eye, and "loss of pep."

While on military service he increased his weight from 130 to 175 pounds, and during the last year has advanced to his present weight of 194 pounds.

In December, 1922 he noticed that the vision in his left eye was failing, and this has advanced to almost complete blindness at the present time. Headache has been present since 1917, but during the past year has been intense. It is bitemporal in location, but recently has involved the occiput as well.

He has never shaved, and, in passing, remarks that his father began to shave when twenty-three years of age. During the past three years he has lost initiative, is easily fatigued and, as he states, "has lost all his pep." He cannot concentrate effectively, continually forgets important missions, and is keenly conscious of a state of inadequacy. He denies venereal disease. Has never vomited.

Examination shows the outstanding feature to be one of adiposity. The fat is generally distributed with marked preponderance about the hips and breasts. The breasts are full and globular, the hips rounded and broad, while the thighs gradually tapir off toward the knees—the general conformation of the female.

The skin is of the "peaches and cream" variety. He has a complexion that any active healthy school girl would envy. You will note that he is almost hairless, for with the exception of the scalp and a downy pubic fringe he is devoid of hair.

The penis is strikingly undersized and the testicles suffer in comparison with a healthy lima bean.

When he speaks it is with the voice of a child and, in effect, his whole bearing suggests that of an unobtrusive, tractable child.

Neurologically, the cranial nerves, with the exception of the optic, are normal.

Pupils are equal, regular, react to light and accommodation.

There is no nystagmus and no ocular paresis.

Examination of the fields of vision shows a bitemporal hemianopsia, fully developed on the left side.

Ophthalmologically, the disks show primary atrophy, more marked on the left side.

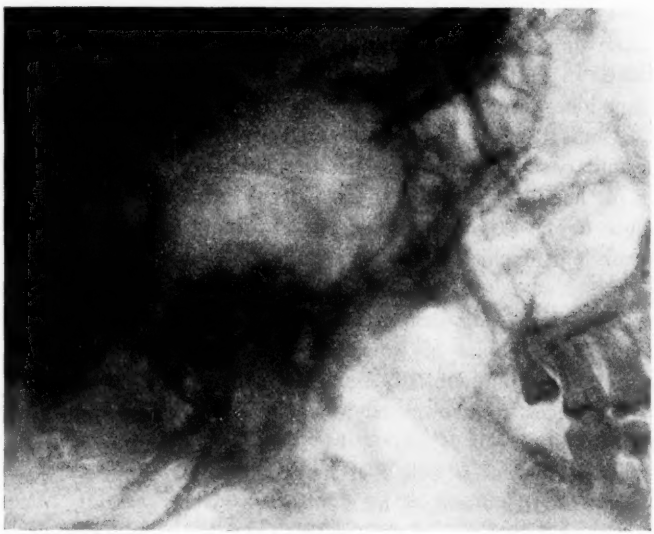

Fig. 305.—Showing extensive destruction of the sella turcica with erosion of the clinoid processes and body of sphenoid. Taken from Case II.

The ocular movements are normally full in all directions.

There is no involvement of the pyramidal tracts as shown in the absence of motor paresis or pathologic reflex activity.

Wassermann reaction is negative.

Basal metabolic rate is shown as —9. Vital capacity —31 per cent.

Urine acid, specific gravity 1022, no sugar, no albumin.

After ingestion of 100 grams of glucose, no glycosuria was observed. Blood-pressure is 115 systolic, 75 diastolic.

Temperature during two weeks' observation in the ward did not rise above $97\frac{2}{5}°$ F.

Radiographic examination shows marked increase in the size of the sella turcica, with erosion of the clinoid processes and the body of the sphenoid (Fig. 305). A further note is that numerous small calcareous bodies are noted in the region of the pineal gland.

This case clearly illustrates the "hypogenitalism" characteristic of decreased pituitary secretion. The evidence contained in the above findings points to decreased function in all three lobes.

Clinically: Dystrophia adiposogenitalis (Fröhlich syndrome) secondary to tumor of the pituitary, probably cystic adenoma. It further illsutrates the effect of local destruction which takes place in the neighborhood of the growing tumor—in this case involving the chiasm.

Case III.—This case is one which is not so easy to classify and which admits of considerable difference of opinion. It is one which undoubtedly finds its place among the endocrine disturbances, and, obviously, bears the markings of pituitary hyposecretion.

It is that of a girl, fourteen years of age, whose present weight is 176 pounds. The adiposity, which is certainly abnormal, is equally distributed over the limbs and trunk. Naturally enough, it adheres to the female conformation.

The mother tells us that she has always been a bright, active, and abnormally strong girl, that she ranks among the leaders in the classroom and on the playground, that fatigue, to her, is almost an unknown quantity, that she has not yet menstruated, that she never loses her temper, that she is most amenable to discipline, which is rarely necessary, and that she is known to all her acquaintances as the "good baby."

Although excessively fond of sweets, she never cries for them or shows resentment if refused them.

Her voice is that of a normal school girl, considerably deeper in pitch than the average. She has a luxuriant growth of soft glossy hair on her head. The skin is soft, moist, and clear, and over the buttocks and shoulders is puckered and mottled.

Her general bearing is that of a self-possessed, self-sufficient vigorous school girl.

Neurologically there are no abnormal findings.

Cranial nerves are normal, fundus is normal.

Motor, sensory, and reflex systems are all normal.

Muscles are unusually strong, and the hand grip is equivalent to that of a strong healthy boy.

Urine is normal. Ingestion of 100 grams of glucose shows no subsequent glycosuria.

Basal metabolic rate is plus 8. Blood-pressure 120/80.

Red blood-cells count 5,400,000. Hgb. 95 per cent.

x-Ray shows a small, rather deep sella turcica, with no evidence of erosion, and normal, well-formed clinoid processes. The abnormal adiposity, coupled with increased tolerance to sugar, certainly suggests pituitary hypofunction, while, on the other hand, the history of activity, muscular strength, and mental acuity distorts the picture of dystrophia adiposogenitalis, a history which our examination corroborates.

What, then, is the explanation of this case? It is shown, here as a case exemplifying the interrelation of the endocrinous glands—in this case, the pituitary and adrenal.

The clinical evidence points to a primary insufficiency of the pituitary with an overcompensatory reaction on the part of the adrenal.

We offer this explanation on the ground that pituitary dysfunction alone will not account for the clinical picture. In fact, our only indication that the pituitary is at fault is found in the increased weight and adiposity, associated with a narrow, deep, and unusually small sellar cavity.

The other side of the picture is one of increased activity of the adrenal cortex which expresses itself in the high level of mental and physical efficiency so characteristic of this patient. Corroborative findings are found in the luxuriant growth of

hair on the scalp and pubis (in the latter case still of the female distribution), the deeply pitched, assertive voice, the general attitude of self-assurance, and the tendency to polycythemia.

If we are right in the assumption that this case illustrates a primary pituitary insufficiency with secondary adrenal hyperactivity, what is the future state of this patient likely to be?

Pathologically, we must assume that a hyperplasia of the adrenal cortex exists. Such an assumption admits of but one opinion.

The female markings, as we see them now, will gradually take on the male characteristics; the hair will become generalized in its distribution, and in the region of the pubes, already overluxuriant for her age, will conform to the male arrangement with a triangular shaped extension upward, toward the umbilicus.

Further accentuation may be seen in the appearance of growth over the chest and upper abdomen, even, in extreme cases, on the upper lip.

The patient becomes more and more assertive and self-reliant and finally assumes a mannish bearing which, in the expression of her unwarranted muscular strength, robs her of the last vestige of the female touch. The final stage of addisonism may or may not appear, but, in general, the ultimate history of these cases is recorded in such terms as "loss of weight," "fatiguability," and "mental inertia," which mark the passing of the pathologic process from hyper- to hypo-activity.

Case IV.—This case is peculiar in that it presents no clinical evidence of endocrine disturbance, though it is of interest from the neurologic viewpoint.

A young man, twenty-seven years of age, showing no apparent signs of endocrine disturbance, presents himself at the clinic for relief from his headache. Like two of the preceding cases he is an ex-soldier, having served two years in the flying corps. During the last year of his service he began to suffer from headache, which, I happen to know, was looked upon as psychogenic in origin. The headache has steadily increased in intensity and,

during the past year, has been constantly present. At the present time it is bitemporal in type, though for the first two years it was confined to the frontal region, with occasional paroxysms over the occiput. During the summer of 1922 his vision began to fail, at first on the right side, but within nine months both eyes were noticeably failing. In February, 1923 he suffered, for the first time, from diplopia, and for several months this "double vision" was most distressing. Because of it he gave up reading, and by the time it had disappeared, in June, 1923, he was unable to read on account of the failure in his vision. In September, 1923 the right upper eyelid began to droop and gradually assumed its present state of complete ptosis. He makes the statement that life would be worth living if he could find relief for his headache, but, as it is, he has, on several occasions, considered suicide, and expresses the wish that the good old days of aërial fighting had not ended. Vomiting occurred on one occasion only, and this he attributes to the taking of too much aspirin. He has given up drugs, as he finds they disturb his stomach and give no relief from his pain.

The family history is devoid of interest.

Examination shows a well-developed, well-nourished man of normal station and gait, weighing 154 pounds, and measuring 5 feet, $9\frac{1}{2}$ inches in height.

From the endocrinologic viewpoint he would appear to be normal. The hair, skin, and subcutaneous tissue show no variation from the normal.

The external genital organs are well developed, especially the testicles, which, one notes, are above average size. The hair distribution about the pubis is definitely of the male type, extending well upward toward the umbilicus. The voice is normally deep and he speaks and acts with the assurance characteristic of his age and sex.

Neurologically we note that the right eye is closed, with complete ptosis of the upper lid. The external ocular muscles on the right side are completely and entirely paralyzed, the eye being incapable of movement in any direction. The right pupil is widely dilated and does not respond to stimulation of

the retina with strong light. Vision is absent in the right eye. On the left side vision is recorded as 6/60. There is apparent limitation of inward rotation of the eyeball, all other movements being normal in range. The pupil is regular, moderately contracted, and reactive to light stimulation. There is no ptosis.

Vision is absent on the right, while on the left the temporal half of the field is markedly contracted.

Ophthalmoscopic examination shows bilateral optic atrophy of the primary type, apparently complete in the right eye.

Fig. 306.—Showing extensive destruction of the sella turcica with erosion of the walls and sphenoid. Taken from Case IV.

Other cranial nerves are normal.

The absence of motor paresis in the limbs and the absence of any form of pathologic reflex activity precludes the possibility of pressure upon the pyramidal tracts from backward extension of the growth.

Urine 1020, acid, no albumin, no sugar by Fehling's test. Ingestion of 100 grams of glucose was not followed by glycosuria.

Basal metabolic rate, plus 8.

Wassermann reaction negative.

Radiographic examination of the skull shows complete erosion of the walls of the sella turcica. The posterior clinoid processes are destroyed, as is also much of the sphenoid (Fig. 306).

Here, then, is a case which presents evidence of extensive local destruction of subjacent structures. The signs point to complete involvement of the right optic tract and, to a lesser extent, the left, while all the motor nerves contained within the right cavernous sinus have been involved in the pathologic growth. The remarkable observation in this case, which is obviously one of pituitary tumor, probably suprasellar, is the extent of local destruction, in the absence of any sign of general endocrine disturbance.

Case V.—The next case to be considered is one of a married woman, forty years of age, who, a few months ago, complained bitterly of headache, loss of interest, weeping spells, and impairment of memory.

For the past twelve years she has suffered from headache and facial pain, which, more recently, has been attributed to her teeth. Extraction followed extraction without the desired relief. During the menstrual periods, which have always been irregular and difficult, she has suffered intensely from the headache and "face pains," the latter being distributed over the right face entirely. Frequently she would experience a dull boring pain in the right eye, which during recent years would culminate in outbursts of profanity, directed chiefly at her husband. When free from the pain she spoke of her husband in the highest terms, and has stated that, were it not for him, she would have ended it all years ago. The headache is bitemporal, but at the height of the paroxysm, which coincides with the menstrual period, it is all over the head. She points to the temporal region, stating "it is just in there."

Her family has always regarded her as nervous, and she believes that her early protected life is responsible for her irritability. She was pregnant once only, and this was terminated

on the advice of her family physician, who considered her too nervous to bear children. She loves her home and states that her happier hours are spent in cleaning it, a recreation she always indulges in when free from pain. Accompanying the more severe attacks is vertigo, which causes the room to reel about her, and from which she obtains relief by lying down.

The family history is not very reassuring. Her mother, always a nervous woman, suffered from headache all her life. Her father was a strong, industrious, and overconscientious man, who knew little else than work. His brother died in an asylum and a cousin was considered "queer" by his friends.

Examination shows a well-developed, well-nourished woman, who measures 5 feet, 7 inches in height and weighs 157 pounds.

On casual observation the case presents no outstanding feature that can be attributed to endocrine dysfunction, but on closer scrutiny the broad forehead, with prominent supraciliary ridges and deeply set eyes, the square broad chin, and apparent decision with which she carries out all her movements, are at least suggestive. The skin is moist, very clear, and remarkably free from lines. The hair is moist, abundant, and of normal distribution. There is apparently normal proportions between the hands, feet, and trunk, and if we observe the relative proportion of the limbs and trunk, it, too, would appear to be within the normal.

Mentally she is bright, alert, and most willing to render assistance in the portrayal of her case. Six months ago she would sit in the chair, refusing to discuss her condition because "after all, it was useless," "everything had been done and she was nothing but a walking drug store."

Neurologically there is nothing of interest in the case.

The cranial nerves are normal, the pupils are equal, regular, and show normal reaction to light and accommodation. There is no nystagmus and no ocular paresis. The fundi are normal. Examination of the fields of vision suggested a slight contraction in both lower temporal quadrants, but this was not definite to the oculist who took the fields.

Motor, sensory, and reflex systems show no variation from normal.

Wassermann, in the blood and spinal fluid, is negative.

Urine is negative to pathologic findings.

Spinal fluid clear and under normal pressure—10 c.c. of Hg. There were 4 cells (lymphocytes) to the cubic millimeter. Globulin could not be demonstrated by the Pandy or Noguchi methods. Wassermann negative.

Basal metabolic rate is plus 6. Blood-pressure 155/120. Radiographic examination of the skull shows a small, covered-

Fig. 307.—Showing closed-in sella turcica with central clouding, suggesting calcareous change. Taken from Case V.

in sella turcica, with no evidence of erosion of the walls. The clinoid processes are intact. In the center of the sellar space is a small area of cloudiness, which suggests calcareous change (Fig. 307).

The point of interest in this case is one of treatment. She was given whole desiccated gland, and almost from the first administration experienced her first real relief from her headache. She has remained free from headache ever since, over a period of four months.

This is a case of headache which, I believe, is due to pituitary dysfunction—a belief which finds support in the immediate relief incident upon gland feeding.

Clinically: Pituitary headache.

It will be noted that this case presents our first attempt at treatment, and I think it may be opportune to say a few words about the treatment of these cases as a whole.

Unfortunately, it requires but a few words to sum up the treatment of pituitary dysfunction other than by surgical means. One would think that regulated feeding of prepared gland substance to the cases which show lowered secretory activity should be productive of much benefit. This, however, has not been the case, and we are, so far as I know, quite as helpless in the medical treatment of dystrophia adiposgenitalis as in allaying the progress of gigantism or acromegaly.

In the treatment, however, of the pituitary headache, as exemplified in the last case, we have had sufficient success to engender the hope that the future may disclose a method of gland feeding which will be equally successful with that employed at present in the treatment of hypothyroid cases.

CLINIC OF DR. J. A. NUTTER

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A STUDY OF SCIATIC PAIN

THE subject of sciatica, or pain from any cause in the distribution of the sciatic nerve, is of great antiquity and corresponding vagueness. It is at best the name of a pain and not the name of a disease. It has, therefore, no standing in purely scientific nomenclature, but on account of its striking and, as it were, challenging feature—chronic and often agonizing pain in the leg—it is convenient to study this pain, its causes and treatment, under its own name. The fact that in most cases this is a referred pain and not due to actual injury to or disease in the nerve itself, was gradually becoming recognized up to the end of the last century. At this time Gowers, of London, dominated the world of neurology, and his dictum that sciatica was essentially a neuritis put an end to fruitful research and restricted treatment to a profusion of drugs and of local applications. Gowers' theories as to its pathology and treatment were copied from text-book to text-book, and may be found even today. Of late years, however, it has become more and more recognized that Gowers was wrong, that it is extremely seldom that actual lesions of the sciatic nerve itself exist, and that nearly all our cases are to be traced to some disturbance in a nearby joint to which the sciatic nerve sends a filament. In this way the doctrine of referred pain is coming into its own, and with it a great strengthening of our hands as regards its relief.

Let us glance for a moment at the anatomy of the sciatic nerve and its surroundings. It arises just above and below the promontory of the sacrum, and is the direct continuation

of the lower two lumbar and the upper three sacral nerve roots. Connecting filaments link it up with other important nerves in the vicinity. It supplies sensation to the lumbar articulations, so that one may easily understand how an arthritis here may express itself in pain down the leg. In addition, malignant disease, usually sarcoma, as well as injury and tuberculous abscess, may, though this is not often seen, press directly upon the nerve roots, causing pressure neuritis. At times the lumbar transverse processes may by fracture or congenital deformity irritate the roots, as these lie directly in front. The upper part of the nerve descends into the pelvis in front of the sacro-iliac joint, supplying it. As this joint is notoriously unstable as compared with the spine or the hip-joint, it is not difficult to understand how injury or disease here is frequently manifested by pain along the sciatic. The nerve is assembled in the pelvis from its various roots, and here is occasionally subjected to pressure from malignant disease or abscess. It leaves the pelvis to enter the buttock by way of the great sacrosciatic foramen, the bony margin of which was once thought, though probably erroneously, to be a frequent source of nerve pressure. As the nerve descends it lies back of the hip-joint, which it supplies. Disease or injury of this joint, together with tumors of this region, may cause nerve irritation. It is to be noted, however, that while a hip arthritis may cause referred sciatic pain, a hip-joint stiffened by old disease or injury is likely to cause sacro-iliac strain, which latter will be the immediate cause of sciatic pain. In the thigh the nerve lies under the hamstring muscles, which it supplies, and where it is at times pressed upon and irritated by inflammatory adhesions of obscure origin, with eventual traumatic neuritis. Usually while in the thigh the nerve divides into two branches, which between them supply all the muscles below the knee and nearly all the sensation. Typical sciatic pain is, as a rule, complained of in the buttock, the back of the thigh, the back of the knee, and the outer side of the lower leg and foot. The pain does not necessarily follow the whole tract, and may even be confined to below the knee. Summing this up, one may say that in the great majority of cases

the exciting cause is either the lumbar spine, the sacro-iliac, or the hip-joint, singly or in combination. The rare cases of direct nerve pressure are nearly always due to malignant disease, abscess of the spine or pelvis together with adhesions in the thigh and deformities of the lumbar transverse processes. One may readily understand how the treatment must vary according to the source of the pain. Gonorrheal arthritis of the spine in one patient and sacro-iliac strain in another may both be accompanied by sciatic pain, but there the resemblance ends. The gonorrheal arthritis will be cured and the sciatica will subside by attention to the urethral tract, while the case of sacro-iliac strain will need a corset and perhaps arch supports. It will thus be seen that successful treatment means a very careful search for the exciting cause and the removal of this when possible.

Let us now consider by what signs or symptoms the source of sciatic pain may be determined. Under what circumstances should the sacro-iliac joint be suspected, the hip-joint, or the lumbar spine, for nearly all our cases are caused by one or more of the three? Let us begin with the sacro-iliac joint, which seems most often at fault.

Lesions of the sacro-iliac as well as of the other joints already mentioned are either traumatic or due to disease, most often an arthritis due to focal infection. In almost every case we are dealing with injury or disease, or both together. Fortunately there are characteristic differences which are of great assistance in arriving at a diagnosis. In any given case of sciatica how is the blame to be laid at the door of the sacro-iliac joint? It is well known that the *x*-rays do not help us here, except perhaps in cases of tuberculosis or malignant disease. Fortunately, we have pain and tenderness at or near the posterior superior spine, localizing evidence which is almost always present and is, therefore, of great assistance. Raising the leg with the knee extended puts strain upon the sacro-iliac joint by pulling against the ischium through the medium of the hamstrings, and is, therefore, likely to increase the pain. When this maneuver succeeds in producing or exaggerating the pain at the posterior

superior spine it is further evidence of sacro-iliac disturbance. At the same time it must be remembered that by this elevation of the extended leg the sciatic nerve is stretched, hence in cases where the pain is severe the test cannot be employed. Strapping, bandaging, or corseting the pelvis is a therapeutic test of great value in suspected sacro-iliac strain, as in the latter condition it generally brings speedy relief. In sacro-iliac subluxation, on the other hand, or in arthritis, especially that due to tuberculosis, this measure may, on the contrary, exaggerate the pain. In a case of the latter disease there will be definite swelling as a rule. Thus in a case of sciatica, with pain and tenderness at the posterior spine which is exaggerated by straight leg raising and greatly relieved at once by strapping or corseting the pelvis, one is justified in suspecting that the sacro-iliac joint is strained and that this strain is the cause of the sciatic pain.

The joint itself is comparatively weak and unstable. A flat-foot, for example, often causes it to ache, as also a short leg or a stiff hip. During pregnancy the weight of the uterus often pulls upon and irritates it, while after parturition it may be left weak and strained by the passage of a large head. The joint has nothing at all comparable to the strong articular processes of the lumbar spine or to the ball and socket of the hip-joint. Treatment obviously means support as well as removal of the exciting cause, if possible. The following brief résumé of a case report will serve to illustrate:

Case I.—A tall, rather heavily built young woman, on her feet a great part of the day, has gradually developed sciatic pain which prolonged rest in bed has not cured. An examination of the patient standing reveals a rather heavy abdomen, tenderness at the posterior superior spine, pain down the back of the left leg exaggerated by straight leg raising, pronation of both feet, but especially the left, no other pain or evidence of arthritis. A corset was fitted, lacing low and in front, with some extra bones in the back, and a pad about the size of the palm of the hand and as thick pressing against the affected

posterior superior spine. This therapeutic test gave her great relief, and a pair of Whitman braces for the feet, aided by baking and massage, completed the cure. The diagnosis was sacro-iliac strain brought on by flat-feet and a heavy abdomen.

Case II.—A middle-aged man with an ankylosed hip-joint suffers from pain in the back and down the affected leg after spending an hour at his desk, at which it is found that he sits with his knee extended. The pain goes away after standing or walking about for some time. It is noted that the position assumed by him at his desk throws a strain upon his sacro-iliac joint, and tenderness is elicited at his posterior superior spine. The diagnosis is made of sacro-iliac strain, and he is advised, with subsequent relief, to use a higher desk and to sit with his knee bent.

When a sacro-iliac joint is strained too often or too much it may subluxate, one of its surfaces twisting a little on the other, and remaining twisted. This usually takes place when the back is bent, so that the upper pole of the sacrum probably moves backward and the pelvis forward. This condition often follows heavy lifting or stooping, though not necessarily, and the patient finds himself (one does not often see this condition in women) unable to straighten up and forced to maintain a stooping position. The interesting point about it is that without a definite history of sudden onset following heavy lifting or other trauma to the back one is almost certain to make the diagnosis of arthritis of the lumbar spine or perhaps lumbago. The patient is forced to maintain a stooping position, the lumbar spine is rigidly fixed, generally at least flat, and not seldom with its curve reversed, its convexity directed backward instead of forward. The whole picture points to the lumbar spine and sacro-iliac displacement is masked. One, however, notices tenderness at the sacro-iliac joint, the absence of evidences of arthritis elsewhere, the sudden onset with trauma, negative *x*-rays, and the absence of relief from rest in bed. The treatment of sacro-iliac subluxation is to restore the joint to its normal condition, and for this an anesthetic is usually necessary.

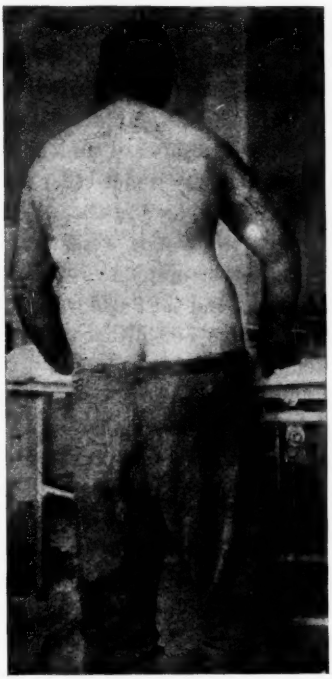
At times the condition reduces itself with a little manipulation, but, as a rule, the strong muscles of the back hold the parts firmly in their distorted position. Under the relaxation of ether the lumbar rigidity disappears, and after hyperextending the legs a few times with counterpressure against the sacrum one applies a plaster spica with the spine well curved in hyperextension. The patient, as a rule, has many complaints to make, both of his new position and of the spica, but in a few days is forced to admit that his sciatica has disappeared. A couple of weeks after the manipulation, and when he has been up and about a few days with no return of the pain, a pelvic support replaces the plaster-of-Paris spica, to be worn for several months. In the case of a man this is made of duck or canvas, lacing in front and kept down by perineal straps so that it embraces the great trochanters. A pad is applied over the affected joint.

Case III.—A medical student, a member of the basketball team, gradually developed a pain down the right leg, and with it a stooping position which he was unable to overcome. The sciatic pain was practically constant, though not agonizing. After some months he gave up the hope that it would disappear spontaneously, and sought relief. The lumbar curve was flattened out and was held rigidly by muscular spasm, while the body was held in a forward position and sacro-iliac tenderness was present. The *x*-rays were negative and there were no pains elsewhere, nor could any cause be found for arthritis in throat, teeth, or genito-urinary tract, etc. The feet were strongly made. In spite of the absence of definite trauma and considering the fact that the patient had subjected himself to a good deal of strain while playing basketball, it was thought possible that sacro-iliac subluxation existed. Under complete ether relaxation the thighs were forcibly hyperextended with counterpressure against the sacrum, a plaster spica then being applied in the position of strong hyperextension of the lumbar spine. After a few days of discomfort it was found that most of the sciatic pain had disappeared. In about two weeks a sacro-iliac support was substituted for the plaster, the pain having by now

disappeared, even though walking had been permitted while wearing the spica. The student reported some weeks later that he was quite free from pain except an occasional twinge when he stooped forward, while the unnatural stooping position had completely disappeared. He will wear the support for some months and will abstain from basketball for the rest of the season.

Case IV.—A 300-pound blacksmith was thrown violently to the ground as the result of a collision, injuring himself so severely

Fig. 308.—Case IV. Sacro-iliac subluxation.



Case 309.—Case IV. Sacro-iliac subluxation.

that for some months he could not walk. He had almost constant sciatic pain dating from the accident. His case being vari-

ously diagnosed and consequently neglected, it was eleven months after the accident that he presented himself to the Orthopedic Out-patient Clinic, walking with difficulty on crutches, as he was a very heavy man. He stood with his body bent forward and away from the affected side (the right), and it was noted that his lumbar spine was firmly fixed, with the curve prominent backward instead of forward, and showing a good deal of rotation forward on the affected side. Some tenderness was present in the region of the right posterior superior spine, and he was quite unable to change the attitude which had existed, so far as he could state, since his accident. *x*-Rays of spine, pelvis, and hips were negative in all respects, with special reference to fracture or displacement. Sacro-iliac subluxation was suspected and the man admitted to the wards, where after a few days' rest he was able to stand more erectly, but still with a definite tilt forward and to the left. On manipulating him in the usual way under ether a definite crepitus was both heard and felt somewhere in the region of the lower part of the back. Several roller towels were then tightly bound about his pelvis and a couple of pillows were placed under his lumbar spine to hyperextend this. He was so heavy a man that he was manipulated in his bed rather than on an operating-table, while no attempt was made to apply a plaster spica. No pain was felt by him in the sciatic region from the time he awoke from the ether, though the lumbar region felt somewhat sore. The freedom from pain has persisted and his body is now straight. A pelvic support is being made, and when this is ready he will be allowed up.

Other sacro-iliac conditions to be considered are tuberculosis and the usual acute and chronic arthritides due most commonly, it would seem, to gonorrhea and tonsillitis. Tuberculous arthritis, as a rule, presents no difficulty except at its onset, before the appearance of swelling and *x*-ray changes. In tuberculous arthritis the sacro-iliac joint should be ankylosed if possible. In the case of the non-tuberculous arthritides one must, if possible, find the cause and remove it.

Case V.—A young man was seen in consultation suffering from acute sciatic pain which was of one week's duration. Sacro-iliac arthritis was suspected, as tenderness existed at the posterior superior spine, but as his temperature had advanced to 104° F. some alarm was felt lest a suppurative condition were present. The discovery of reddened tonsils revealed the source of the arthritis, and with their return to normal the sciatic pain disappeared.

The lumbar spine is the most vulnerable part of the vertebral column because it is the most movable and bears the greatest weight. The ribs act as splints to protect the dorsal spine, while the lumbar region enjoys no such protection. In a consideration of sciatic pain in relation to the lumbar spine the arthritides due to tonsil, teeth, urethra, etc., are in the front rank. When present one finds the spine fixed by muscular spasm, and jarring it usually is painful. When the arthritis has been present several years the whole spine may be found stiffened, and characteristic lipping may be shown at the upper and lower margins of the vertebral bodies in the *x*-rays. The condition, as a rule, is easily diagnosed, and the treatment is rest, support to the spine by corset, back brace or plaster spica, baking and massage, and removal, if possible, of the cause of the arthritis. It will be found helpful to bear in mind that just as a stiff hip is likely to cause sacro-iliac strain, so also a stiff spine may have the like result. The moral is that when one is treating a case of sciatica in a patient with undoubted chronic lumbar arthritis the possibility, even the probability, of sacro-iliac strain should be borne in mind.

Case VI.—A man of fifty was a constant sufferer from sciatica. His lumbar spine and part of his dorsal spine also were ankylosed as the result of chronic non-tuberculous arthritis (arthritis deformans). He had had excellent treatment so far as concerned the lumbar spine, but without much benefit. As sacro-iliac tenderness was found, a pelvic support was recommended, with great relief to the strain which there existed and to the sciatica which had resulted.

It is instructive to know that chronic arthritis may exist, though unsuspected, for years in a spine, manifesting itself by only an occasional twinge of pain and a feeling of stiffness. Some jolt or jar at length adds the final touch of irritation, and acute pain is felt which shows no disposition to leave.

Tuberculosis of the spine at a level sufficiently low to be a cause of sciatic pain will need a plaster spica for fixation, and, if possible, an ankylosing operation. A tuberculous abscess suspected of nerve pressure should be evacuated by aspiration. Such a condition is uncommon. Malignant disease of the spine may cause intractable sciatic pain, and is, unfortunately, hopeless. Fracture of the spine usually occurs at the dorsolumbar junction, too high to be a direct cause of sciatica, but quite capable by reason of the stiffness of the spine which it causes of throwing a painful strain on the sacro-iliac joints. Even though the pain due to the fracture may be irremediable, that due to the sacro-iliac strain should be curable. When a case of intractable sciatic pain is aggravated by pressure over the tip of a transverse process the question of nerve pressure by this bony process should be considered. Cases are on record where a cure has been obtained by the removal of a transverse process found deformed by old fracture or congenital malformation.

The lumbosacral region is the area par excellence of congenital deformity. "When in doubt play the fifth lumbar" is popular, even though of doubtful utility. Many cases of sciatic pain show congenital anomalies of this region, and the temptation is strong to lay the guilt at their door. It is probable, however, that even these cases suffer from such common ailments as sacro-iliac strain.

Case VII.—A boy of sixteen was admitted to the Montreal General Hospital suffering from sciatic pain without the history of trauma. As he showed an exaggerated lumbar lordosis and had no waist-line in evidence, due to the fact that his lower ribs rested almost directly upon his iliac crests, he was suspected of spondylolisthesis. The *x*-rays confirmed this, showing de-

fective ossification of the laminæ of both the fifth lumbar and the first sacral, the former vertebra being balanced on the promontory of the sacrum and well forward of its normal position. In the presence of this interesting feature such commonplace conditions as flat-feet and sacro-iliac strain were left unconsidered. Before long, however, these assumed their proper importance, and it is likely that by the use of foot-plates and a support for the pelvis and the lumbar spine he will obtain relief. He has commenced treatment only recently, and has been warned to avoid strenuous games on account of his weak back, which needs only moderate violence to break in two.

At the same time it must be admitted that cases of undue mobility at the lumbosacral junction exist, probably the result of congenital deformity here. In such cases one may expect direct nerve pressure, with paresis or paralysis of the leg muscles, together with pain along the tract of the affected nerve.

Disease or injury of the hip-joint is frequently a cause of sciatic pain. The condition is only too often and apparently quite satisfactorily diagnosed as sciatica, while the causative factor, frequently tuberculosis of the joint, remains unnoticed.

Case VIII.—A boy of nineteen was referred for the treatment of sciatic pain after several months of massage and manipulation. His condition had been diagnosed and treated as a pain, and pain only. His hip movements were greatly restricted, the thigh showed atrophy, and in the *x*-rays definite bone destruction was evident. Syphilis being excluded, a diagnosis of tuberculosis of the joint was made, and a plaster spica applied, with rest in bed and open-air treatment. In a few weeks the pain had subsided. Had it been rebellious, traction would have been employed. Errors of diagnosis are more likely to occur in acute than in chronic arthritis, as in the case of the latter joint stiffness and *x*-ray changes become very obvious and can hardly be overlooked. It is scarcely necessary to state that an arthritis will show limitation of movement in all directions, while a normal hip-joint in the presence of sciatic pain will be restricted

in those movements only which stretch the sciatic nerve, notably flexion of the hip with the knee extended.

Gonorrheal arthritis of the hip, as a rule, develops acutely and very painfully. Here also the diagnosis is apt to be sciatica unless the development of arthritis in other joints and the known presence of gonococcal infection make the condition clear. The patient, as a rule, is unable to move on account of the severe pain, for the relief of which sedatives will be needed to supplement immobilization and traction. It is interesting to note that the *x*-ray picture of gonorrheal arthritis during the first few weeks may be not unlike that of tuberculosis, with rarefaction and beginning destruction of the articular cartilage. It not infrequently happens, therefore, that a supposed tuberculous hip-joint recovers with extraordinary rapidity. Later on, of course, the *x*-ray findings become very characteristic, the bony proliferation at the acetabular margin due to gonococcal arthritis contrasting unmistakably with the well-marked bone destruction of tuberculosis. The sciatic pain with which the arthritis often begins before long overspreads these limits and becomes general on all aspects of the hip-joint. Only too often a firmly ankylosed hip follows gonorrheal infection.

Syphilis of the hip-joint may be a cause of sciatic pain, and, though uncommon, should be borne in mind.

Case IX.—A young woman, a shop employee, was forced to leave her work one day suffering from pain in the hip and back of the leg. No trauma had occurred, no cause was known. For the next six weeks she was treated at home for sciatica by sedatives and liniments, etc., until, on account of the increasing pain, she could with difficulty stir. Two months after the first appearance of the pain she was admitted to the Montreal General Hospital, her hip in extreme adduction and flexion. Her condition was thought to be one of acute tuberculous arthritis until the *x*-rays revealed extensive destruction at the hip-joint, both of the femoral head and of the acetabulum, with pathologic dislocation. Such a condition could have been attributed to tuberculosis if only of more than a year's standing,

while the history of her illness dated back precisely two months. A gonorrheal arthritis was ruled out by the low temperature, speedy relief from pain by plaster fixation, and the absence of other joint involvement. The rapidity of bone destruction was understood only when a markedly positive Wassermann revealed the presence of a syphilitic arthritis. She rapidly recovered under appropriate antiluetic medication and a couple of months later was able to walk about unaided in her spica, her hip ankylosed, and her leg $1\frac{1}{2}$ inches short. The deformity at the hip had been reduced as far as possible under ether.

In patients over forty a chronic non-tuberculous arthritis (arthritis deformans) of the hip may be a cause of sciatic pain. Its origin is too often undiscoverable. This disease is much more common in England than in America, and, in fact, one English physician wrote a whole book to prove that all cases of sciatica are from this source. The disease is painful, chronic, and often very difficult to relieve. The *x*-rays show destruction of the hip-joint with bony outgrowths at the lip of the acetabulum. Movements of the joint are painful and weight bearing frequently impossible, and it is often that the pain disappears only as the joint becomes ankylosed. In severe cases rest in bed with plaster fixation and perhaps traction may bring relief, while in others pillow fixation is assisted by baking or hot applications. A Thomas splint makes walking possible without weight bearing, a high sole being worn on the good foot. In cases where but little movement is left, and that is productive of pain, an ankylosing operation may be performed at the hip with relief.

It has been shown that in the vast majority of cases sciatic pain is the complaint of a joint that is suffering, the suffering being due to either mechanical disability or to infection, or, as often happens, to a combination of both of these. It is this last, joint strain combined with arthritis, that may now be considered, for it frequently occurs, and upon its recognition successful treatment depends. A flat-foot may appear to be a flat-foot pure and simple, but if, in addition, a gonorrheal arthritis is present and is not recognized and treated, the result

of prescribing a foot-plate will not be a happy one. Precisely in the same way a case of supposed sacro-iliac strain may fail to respond to the therapeutic test afforded by a well-fitting corset or pelvic support, indicating, as a rule, that the diagnosis is one of infection rather than strain. It is thus to be seen that the balancing of mechanical strain against arthritis is of the greatest possible importance in considering the problem of sciatica. In difficult cases it may become necessary to eliminate all possible mechanical factors before one can be sure that infection exists, and vice versa.

Case X.—A tall spare bank messenger was admitted to the Orthopedic Service of the Montreal General Hospital, suffering from sciatic pain which had been present on and off for six months, without definite history of trauma. He gave a history of backache the year previous, and this, together with the fact that his lumbar spine was held quite rigidly and that his feet were not flat, was held to be sufficient evidence to warrant a diagnosis of lumbar arthritis, though no other joints were involved. A diligent search was made to find an infective focus to account for the arthritis, and as his tonsils were reported suspicious by the Department of Laryngology, they were removed, but with no effect on the pain. A well-fitting sacro-iliac support gave but slight relief, while various diets, massage, baking, etc., were given in vain. *x*-Rays of the spine had always been negative. On reviewing the evidence in his case it was noted that the lumbar spine curved backward instead of, as is normally seen, forward, while there had always been some tenderness in the region of the posterior superior spine on the affected side. As infection had as far as possible been eliminated without success, it was thought that the condition, in spite of the lack of history of trauma, might be of a mechanical rather than an infective origin. With this new theory in view the sacro-iliac joint was suspected of subluxation, and reduction was accordingly attempted under ether. With complete relaxation the hips were forcibly hyperextended while counter-pressure was made against the sacrum, the patient then being

put in a plaster-of-Paris spica with the lumbar spine hyperextended. Owing probably to insufficient padding and the strained position the patient found a great deal of fault with the spica, though he admitted that the sciatic pain had left every region but the foot. After some three or four days he demanded that the plaster be removed, and a sacro-iliac support was substituted, to his great relief. With some surprise it was noted that he no longer complained of his pain, even when allowed to walk about the ward. The sciatica did not return, and about ten days after leaving the hospital he reported for duty as bank messenger once again. A telephone message a few weeks later gave the assurance that all was well. This case is instructive in that the rigidity of the lumbar spine led to an erroneous diagnosis of arthritis, by which a good deal of time was lost.

In similar fashion a sciatic sufferer with gonorrheal arthritis of spine and feet may have efficient treatment of these regions with but partial relief of his leg pain, owing to the neglect of treatment of his flat-feet and secondary sacro-iliac strain. At times two distinct mechanical factors are at fault, and for relief of the pain both need attention.

Case XI.—A young woman suffered greatly with sciatic pain after parturition, and showed sacro-iliac pain and tenderness. Relaxation of this joint is common, as it partakes in the general hyperemic softening which accompanies pregnancy, as well as being subjected to considerable trauma by the child's head during its passage through the birth canal. Sacro-iliac strain was, therefore, diagnosed and a supporting corset fitted, with considerable relief. On getting up and about the patient seemed unable to obtain complete freedom from pain. Further examination then showed that the woman had a flat-foot on the affected side, the relief of which by means of an arch support caused the final disappearance of her sciatica.

Patients with sciatica should, so far as possible, be examined while standing, as owing to fatigue after a few minutes a weak arch may reveal itself.

Case XII.—A male patient was referred for pain back of the hip and radiating down the thigh. A most careful examination failed to reveal any trouble, infective or mechanical, in spine, sacro-iliac, or hip-joint. No cause of the pain was to be found and no other painful joints, as well as no history of trauma. By this time the patient had been on his feet, which had been bared, for five or ten minutes, and it was now for the first time to be noticed that the arch of the foot on the affected side was definitely weak. The hip and leg pain was at once relieved by the use of a properly fitted arch support, the diagnosis probably being strain of the hip induced by the flat-foot.

In relation to the whole number, but very few cases of sciatic pain stand revealed as due to direct pressure on the nerve. In a few cases lumbar transverse processes deformed from birth or following fracture have been excised with relief to an intractable pain. In such instances the pain, whether in the back or in the sciatic tract, has been aggravated by pressure over the tip of the transverse process. Malignant tumors, as a rule sarcoma, may attack spine, pelvis, or hip-joint. Abscesses may occur along the spine or in the pelvic cavity. The pregnant uterus and the overloaded colon are credited by many authors with nerve pressure in the pelvis, but against this theory there is considerable evidence that these may act by causing sacro-iliac strain. In the thigh obstinate cases of sciatica have been cut down upon, the nerve in some cases being found throttled by inflammatory adhesions of doubtful origin. The nerve has been freed from its adhesions, with immediate relief. Neuromata may attack the nerve, while varicose veins inside the sheath may be a source of pressure. At times a portion of the nerve enters the pelvis between the sciatic notch and the pyriformis muscle, instead of below the latter. In such cases the muscle on contracting is thought capable of exerting injurious pressure on the nerve above it.

Not infrequently one meets with a case of sciatic pain in association with diabetes or chronic nephritis, where under an appropriate diet the pain disappears. It is difficult to decide

into which category such cases should go, whether they are to be classed as neuritic or are referred pain.

One may perhaps close with the plea that cases of sciatic pain be examined thoroughly, the back and affected leg bared and the patient standing, and that a careful search be made for trouble, either mechanical or infective, in the joints known to be capable of being the cause. If this were done more often the name sciatica would lose much of the hopelessness it now inspires, and fewer patients would desert the medical profession for the irregular practitioner.

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MONTREAL GENERAL HOSPITAL

PRIMARY SARCOMA OF THE MEDIASTINUM AND LUNGS

MALIGNANT tumors of the thoracic cavity, like tumors elsewhere, are either primary or secondary. They may be of any type, but the common primary ones are either sarcoma or carcinoma. This clinic will deal only with sarcoma. Growths in the thoracic cavity have been known and described by various writers in the seventeenth and eighteenth centuries. In 1865 Cockle published his book "On Intrathoracic Cancer." In 1892 John Steven published in London a book entitled "The Pathology of Mediastinal Tumors with Reference to Diagnosis." Since that date many papers have been published, but few add much to our knowledge of the subject. Sarcoma tumors in the mediastinum originate from the connective tissues, from the lymph-nodes, and from the thymus. Some consider that the commonest source is the thymus gland or the remains of the thymus gland. Steven stated that they most often arise from the lymph-nodes; he is probably correct. They grow by extension and often mold themselves around the vessels and nerves, and the damage done to these structures is much more often due to pressure than to actual invasion of them. They seem to grow in the direction of least resistance. Steven was the first to note this, and most writers since agree with his observations. In various series tabulated by Christian,[1] Hebert,[2] and others by decades the numbers were about equal, from 20 to 60. A few cases occur before and a few later. In this series 8 out of the 13 cases occurred in the fourth and fifth decades, with 2 in the third, 2 in the seventh, and 1 in the first.

The symptoms of onset are usually gradual, and only occasionally does one find them coming on abruptly:

1. Dyspnea on exertion is an early symptom, and was present in 100 per cent. of this series.

2. Cough and expectoration are usually present. The amount of expectoration is scanty, and was blood tinged or bloody in 50 per cent. of this series.

3. Sense of tightness in the chest was complained of in some of the cases.

4. Pain in the chest is a common symptom and varies from discomfort to very severe pain, often described as darting in character. With involvement of the pleura it becomes of the pleural type and is increased by coughing and deep breathing.

5. Night-sweats occurred in 25 per cent. of the cases.

6. Fever of a moderate grade, often of the remittent type, was present in 75 per cent. of the cases.

7. Loss of weight may not be an early symptom, but is present in all cases sooner or later.

It will be noticed that all the above symptoms are common in pulmonary tuberculosis, with which this disease is often, at first, confounded.

Other symptoms were present in some of the cases, due largely to pressure, viz., cyanosis, dilatation of veins, edema, hoarseness, dysphagia, and unequal pupils.

The symptoms may not be aggravated for weeks or months, but tend to get progressively worse.

The *physical examination* shows sometimes prominence of the upper portion of the chest and a tumor in the suprasternal notch. Localized cyanosis, edema, and dilatation of veins of chest, neck, and arms are commonly seen.

Dulness underneath the upper portion of the sternum is present in tumors of the upper mediastinum, but not in tumors of the posterior mediastinum. When the tumor is in the lungs there is diminished expansion and dulness on percussion on the affected side. Breath sounds, as a rule, are diminished, but may be distinctly blowing in character.

Râles of various types are invariably present. Pressure

signs may also be present, such as from pressure on the left recurrent laryngeal nerve, giving immobile vocal cord and characteristic hoarseness, on the trachea to stridor, on the sympathetic to altered pupils, on the esophagus to difficult swallowing, etc.

It is not always possible to tell clinically if the sarcoma arises primarily in the mediastinum or in the lungs unless the case is seen early. Undoubtedly, primary sarcoma of the mediastinum is more common than primary sarcoma of the lungs.

The *x*-ray is a most important aid in the early diagnosis. Probably the reason why it is so difficult to tell whether they are primary in the mediastinum or the lungs is due to the fact that they quickly grow, so that all the structures in the thorax may become involved. While it is true, as previously stated, that they often mold themselves around the neighboring structures, in other cases they grow into them. In some of our cases recorded below we find the disease in the esophagus, heart, pericardium, and blood-vessels. The bones are not often involved.

The tumor may also spread in the chest by metastasis, as well as by direct extension. Metastases occur elsewhere, and if a vein is ruptured they may appear anywhere, as in the liver, spleen, brain, etc.

Diagnosis.—Where there is a tumor of the mediastinum the diagnosis can usually be made. It is sometimes difficult to differentiate from aneurysm. The absence of pulsation, tracheal tugging, and unequal pulses point to sarcoma. If the patient remains under observation for a time there is usually no difficulty in deciding which disease is present.

Where the lung is involved *en masse* the physical signs resemble somewhat those of fluid in the pleura, and in many cases fluid is present. The dulness is very marked and the breath sounds diminished.

Paracentesis shows almost invariably bloody fluid, in large amounts if from the pleura, blood-stained if the lung tumor is pierced. In the latter case the needle gives the impression

of entering a solid medium. In exceptional cases the fluid may not be bloody.

Treatment.—Case V showed marked improvement for a time by *x*-ray treatment. It demonstrated definitely the remarkable disappearance of the superficial portion of the tumor, and suggests the possibility of cure from this form of treatment, when it can be employed as efficiently more deeply.

One case was treated by radium, without benefit.

Duncan[3] reports 3 cases treated by radium—2 cured and 1 improved.

No surgical treatment was used in this hospital, and when one considers the postmortem findings one feels that surgery offers no hope of successfully dealing with the disease.

Case I.—J. S., male, aged thirty-four. Admitted November 21, 1920. Died February 14, 1921.

Complaints.—Pain in left side of chest. Cough with expectoration.

Personal History.—Contracted syphilis 1918. Uses tobacco and alcohol moderately.

Family History.—Negative.

Present Illness.—Not been feeling well for two months. About six weeks ago developed a cough with a little expectoration, with occasional thick clots of blood. Stopped work three weeks ago on account of weakness. Pain has not been severe. Cough and expectoration continued.

Present Condition.—Small man. Much emaciated.

Resp. Sys.—Chest is narrow and flattened on left side. On left side there is diminished expansion and flat note in middle axilla and from spine of scapula to base. Traube's space is dull. Breath sounds are diminished, with whispering pectoriloquy over dull area. Grocco's triangle dull. Heart is not displaced. Two days after admission 10 c.c. of bloody fluid removed from left pleural cavity. On staining smear with Wright's stain one embryonic cell, showing typical mitosis, was found. Fluid in chest increased until left pleural cavity was full. Was given antiluetic treatment, without improvement.

January 20, 1921: Face is very puffy. No edema below waist. Upper portion of chest edematous. Second, third, and fourth left costal cartilages are swollen. Veins of upper portion of chest distended. Left femur, middle portion, swollen. Wassermann +++.

x-Ray showed homogeneous shadow of whole of left chest. No displacement of heart.

Illness practically afebrile except for a few days, with slight elevation of temperature. Highest 101.2° F. Gained 3 pounds in weight.

Postmortem Findings.—Sarcoma of mediastinum. Metastases into left femur, left lung, and pleura, ribs, pericardium, left auricular wall, and peribronchial glands.

Hemopericardium.

Case II.—J. G., male, aged sixty-two. Admitted January 15, 1921. Discharged February 3, 1921.

Complaints.—Unable to walk. Pain in left leg above knee. Cough.

Personal History.—Chancre(?) thirty years ago. Neisser several times. Uses alcohol and tobacco to excess.

Family History.—Negative.

Present Illness.—For past year he has had a cough with considerable thick expectoration. Pain over left lung for months, made worse by coughing. Does not think he has lost weight.

Present Condition.—There is an area of dulness in left lower lobe with diminished fremitus, breath and voice sounds. Around the outer and lower margins of this area there are numerous medium moist râles. Needle withdraws small amount of bloody fluid.

x-Ray examination shows a dense shadow in the posterior mediastinum, the inner margin of which cannot be separated from the shadow of the vertebral column. The upper margin is more or less pointed and is on a level with the upper level of the aortic arch. The width of the posterior mediastinum is increased and the heart is evidently displaced forward. There is a metastasis in the middle third of the left femur.

Case III.—D. G., female, aged twenty-six. Admitted January 17, 1921. Discharged January 21, 1921.

Complaints.—Puffiness of face. Enlargement of thyroid gland. Shortness of breath on exertion. Palpitation of heart.

Personal History.—Negative.

Family History.—Mother died of cancer of stomach, aged fifty-five, a year ago.

Present Illness.—Early in December, 1920 noticed face puffy, and that it became congested on bending forward. Has had shortness of breath on exertion for six months and also when talking in the recumbent position.

Present Condition.—Is well nourished. No pain. Eats and sleeps well. Whole face is decidedly swollen. The veins of neck, especially of left side, are distended. Veins of left side of chest are markedly distended, looking more like small varicose veins than simple distention.

Resp. Sys.—Chest: The suprasternal notch is distended. There is an absolutely flat note over the whole of the sternum, and 1 inch to left and right, above the heart dulness. There is a dull note in left axilla and base, with dim breath sounds.

x-Ray examination of chest shows a broad mediastinal shadow, the heart and aorta being indistinguishable. A fairly dense shadow is present at left base, suggestive of fluid.

Patient was treated by radium, without effect on the growth of the tumor, and died two months later at home. No post-mortem allowed.

Case IV.—J. B., male, aged forty-eight. Admitted August 24, 1921. Died September 11, 1921.

Present Illness.—In the autumn of 1920 had a "stuffy sensation" in chest and was short of breath on exertion. Cough commenced in February, 1921, with whitish sputum, once or twice being blood-streaked. Has severe pain over upper part of heart. Loss of weight 8 pounds. Appetite poor. No history of syphilis.

Family History.—Negative.

Present Condition.—Big man. Nutrition good. Color good.

Resp. Sys.—Marked dulness in chest from left apex to second

rib, with bronchial breathing. Crackles heard over rest of upper lobe and into axilla. Dulness underneath upper portion of sternum. Left apex behind is dull, with a few crackles. Voice is husky and gets worse toward evening. There is a complete paralysis of left vocal cord. Pericardial friction-rub a few days before death.

Postmortem Findings.—Mediastinal tumor (sarcoma) invading upper lobe of the left lung, pericardium, and compressing both branches of pulmonary artery and left primary bronchus, and the nerves in the left half of the mediastinum. Tumor thrombus extending from left pulmonary vein into left auricle. Hemopericardium from tumor. Inactive tuberculosis, apex of right lung.

Case V.—P. L., male, aged thirty-seven. Admitted January 3, 1922. Died July 3, 1922.

Complaints.—Swelling on his chest. Cough and expectoration. Pain in right side of chest.

Family History.—Father died aged thirty-nine of tuberculosis. One brother died of tuberculosis.

Personal History.—Syphilis in 1915. Uses alcohol and tobacco to excess. Has lost 15 pounds in weight during last six months.

Present Illness.—Patient noticed swelling over the sternum about eighteen months ago. This lasted two months and almost disappeared. This caused no symptoms. About three months ago swelling began to increase in size, and has grown very rapidly in the last two weeks. Has had a cough for a long time, and this has become much worse. The swelling gives him a sense of constriction in the chest when he walks about, and he is unable to turn on his side in bed on account of tenderness of swelling.

Present Condition.—Well developed and well nourished. Glands: Left anterior cervical glands are enlarged, largest being size of an acorn. Axillary glands enlarged. Largest the size of a walnut.

Resp. Sys.—There is a very large irregular tumor on front of chest (Figs 310, 311). There is dulness over whole tumor mass.

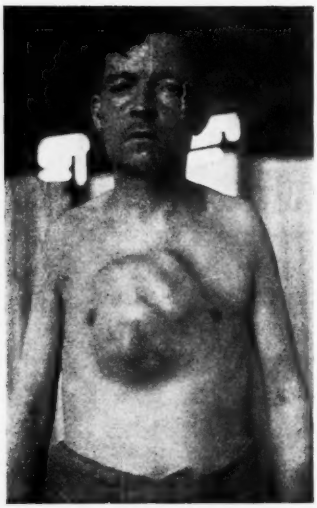

Fig. 310.—Case V. Before treatment.

Fig. 311.—Case V. Before treatment.

Fig. 312.—Case V. After treatment.

Fig. 313.—Case V. After treatment.

Marked dulness from eighth rib to base. The dulness is movable. Breath sounds diminished. There is a loud friction-rub at right base.

x-Ray treatment used, and by February 6th the tumor mass had almost disappeared (Figs. 312, 313).

March 28, 1922: Liver and spleen are enlarged and palpable below costal margin.

May 30, 1922: Liver extends a handbreadth below costal margin. Numerous small subcutaneous firm nodules have appeared over the lower portion of right chest.

June 8, 1922: Evidence of fluid in left pleura. Heart dulness is enlarged. There is a soft systolic murmur and a pericardial friction-rub.

June 12, 1922: Evidence of fluid in peritoneum. Fluid withdrawn from right pleural cavity is blood tinged. Wassermann triple plus. During treatment with *x*-ray, when tumor was disappearing, patient would run a high continuous temperature for ten days or so, during which time treatments were discontinued.

Postmortem Findings.—Lymphosarcoma of mediastinum with invasion of pleuræ, lungs, heart, diaphragm, glands, skin over sternum, liver, mesenteric glands, retroperitoneal glands, and peritoneum. Active tuberculosis at left apex.

Case VI.—L. B., female, aged thirty-four. Admitted June 24, 1922. Discharged September 7, 1922.

Complaints.—Cough. Weakness. Loss of weight. Shortness of breath.

Personal History.—Tonsils removed in 1918. Gall-bladder and appendix removed a year ago in this hospital. Not malignant. No venereal history. Does not use tobacco or alcohol.

Family History.—Mother dead, aged fifty-nine. Pleurisy and jaundice. One sister and two brothers died of pulmonary tuberculosis.

Present Illness.—Following gall-bladder operation patient had phlebitis of left leg and remained in bed two months. Commenced to work as stenographer in February, 1922, but she states

that she has never been strong since operation. Three months ago had a severe bronchitis and was in bed a week. Cough has persisted with a little expectoration, which has been getting much worse during past three weeks. During this time there has been shortness of breath on exertion and now there is dyspnea when in bed. For the past few days there have been knife-like pains in the left chest. Loss of weight—10 pounds in three weeks.

Present Condition.—Is well developed and well nourished. She prefers to sit up in bed on account of dyspnea. Glands normal.

Fig. 314.—Case VI.

Resp. Sys.—Chest is symmetric and upper third of left chest bulges. There are dilated veins on uppor portion of left chest and left arm. There is a dull note from apex to heart on left side, and this dulness extends 1 inch to right of sternum. Behind there is dulness from apex to spine of scapula and from ninth rib to the base. Over the dull area the breath sounds are diminished and there is some blowing, breathing, and bronchophony with a few coarse râles.

July 1, 1922: Complains of dull constant pain between scapulæ.

July 24, 1922: Considerably increased dull area.

September 7, 1922: Patient left hospital in poor condition, as she wished to die at her home in the country.

x-Ray (July 20, 1922) shows marked increase in the width of the central shadow to the left above and to the right below, the outer margins being convex in these regions. The left ventricle is made out clearly, but the aortic shadow is obscured by the projection of the central shadow.

x-Ray (August 21, 1922) shows marked increase in the width of the central shadow, the upper half to the left and the middle third to the right. The free margins of this shadow are more or less convex. The tracheal shadow is displaced markedly to the right. The heart shadow shows slight displacement to the right.

Vocal cords normal.

Febrile during whole course of illness. Highest temperature 101.8° F.

Case VII.—S. W., female, aged twenty-four. Admitted June 18, 1922. Died July 25, 1922.

Complaints.—Weakness. Loss of appetite. Pain in right side. Cough. Feverishness.

Personal History.—Negative. Married at twenty-one. Has baby eighteen months old. No venereal history. Does not use tobacco or alcohol.

Family History.—Negative.

Present Illness.—Illness began on June 9th, when she was awakened about 4 A. M. with a pain in right side. Pain was stabbing in character and worse on breathing. She remained in bed for a day, and the following afternoon went out, although the pain was still present, but not so severe. She remained in bed from the 11th until admitted to the hospital. Pain still present. No cough.

Present Condition.—Well developed and well nourished. Prefers to sit up in bed. Breathing is rapid; lips and finger-tips

cyanosed. She complains of a good deal of pain in right side. Glands negative.

Resp. Sys.—Expansion diminished on right side. Fremitus diminished over right base behind. There is dulness in right lower axilla and from fifth rib to base behind. Over the dull area the breath sounds are much diminished, with distant blowing breathing and bronchophony and a few fine crepitations.

June 21, 1922: Exploratory puncture gave 20 c.c. of fluid almost pure blood.

Fig. 315.—Case VII.

June 23, 1922: 500 c.c. withdrawn, almost pure blood.

June 24, 1922: Dulness extends higher to the second rib. Also under the sternum to the left middle clavicular line. The note at left base is impaired, but not dull.

July 11, 1922: Aspirated, but only 5 c.c. of bloody fluid withdrawn.

July 20, 1922: No fluid obtained.

x-Ray on June 22d shows a fairly dense homogeneous shadow

occupying the lower two-thirds of right chest. Also a less dense shadow in lower portion of left chest, in the upper portion of which is a circumscribed shadow about the size of a 50-cent piece.

x-Ray on June 28th was the same as above.

x-Ray on July 21st was the same as above.

No leukocytosis.

No growth on fluid.

Illness febrile throughout. Highest temperature 103° F.

Case VIII.—M. C., female, aged sixty-three. Admitted June 21, 1922. Died October 11, 1922.

Complaints.—Cough. Shortness of breath. Expectoration blood tinged. Weakness. Loss of weight. Night-sweats.

Personal History.—No venereal history. Does not use tobacco or alcohol.

Family History.—Father died aged thirty-seven, pulmonary tuberculosis.

Present Illness.—Has not been feeling well since June, 1921, on account of loss of appetite and failing strength. Cough developed in February, 1922, and this caused her to give up work; being a domestic, people did not like to have her. The cough was dry and unproductive at first and worse at night. Loss of weight 17 pounds on admission.

Present Condition.—Thin, poorly nourished woman. Prefers to sit up in bed on account of cough and dyspnea. Glands negative.

Resp. Sys.—Left side of chest moves less than right. There is dulness of left chest from apex to heart, dulness extending out to anterior axillary fold. Breath sounds diminished over dull area. Slight blowing and breathing below left clavicle. Resonance normal behind.

July 23, 1922: Dulness with diminished breath sounds from third rib to base behind. Exploratory puncture, but no fluid withdrawn.

August 16, 1922: Exploratory puncture gave a few cubic centimeters of blood-stained fluid.

September 2, 1922: Edema of right hand marked. Right supraclavicular gland enlarged and palpable.

September 5, 1922: Impaired resonance in right apex and evidence of fluid in peritoneum.

September 8, 1922: Right pupil much larger than left.

September 18, 1922: Glands in the right supraclavicular fossa much enlarged.

September 22, 1922: Left side of chest, especially near upper part of sternum, bulges. There is also marked dulness under sternum.

September 26, 1922: Left leg edematous and prominent venules over left side of abdomen.

October 4, 1922: Edema of left hand and forearm. Liver extends 6 cm. below costal border.

October 9, 1922: There is marked stridor. Sputum constantly blood tinged. Many examinations negative for acid-fast bacilli.

September 29, 1922: Left vocal cord paralysis.

x-Ray on June 30th shows presence of dense homogeneous shadow occupying lower two-thirds of left chest. Right hilus enlarged and blurred. Slight cardiac displacement to the right.

x-Ray on August 21st was as above, with slight displacement of trachea and heart to the right.

x-Ray on September 19th was as above. No displacement of heart to the right.

Illness for most part afebrile. Highest temperature 103.4° F.

Postmortem Findings.—A small round-cell sarcoma occupying the whole of the left chest, causing enlargement of the lung and mediastinal lymph-nodes. The left recurrent laryngeal nerve is embedded in the tumor tissue and the trachea narrowed by pressure. There are no metastases outside the chest with the exception of the supraclavicular lymph-nodes of the right side.

Case IX.—I. M., male, aged forty-two. Admitted September 22, 1923. Died October 5, 1923.

Complaints.—Pain between shoulder-blades. Cough and expectoration. Loss of weight. Weakness.

Personal History.—No history of venereal disease. Moderate user of tobacco and alcohol.

Family History.—Negative.

Present Illness.—Began nine months ago with a cough, which was quite severe, with much expectoration, which at that time was often blood-tinged. Recently it was greenish and yellowish. A few weeks after the cough commenced he complained of pain between the shoulder-blades, this sometimes radiated to the front. This pain is more or less continuous and worse at night. Bending forward increases the pain. Breathing does not affect pain. Patient has been in bed since onset of illness. The pain has become more severe and prevents sleep. Loss of weight from 185 to 116 pounds.

Present Condition.—Patient is poorly nourished; eyes sunken. Mucous membranes pale. Very marked clubbing of fingers and toes. Glands: Axillary glands slightly enlarged, others normal.

Resp. Sys.—Chest is symmetric. Left side moves less than right. The left side is dull from apex to fourth rib, and behind from apex to fourth dorsal spine. Also upper portion of sternum. The breath sounds are diminished over dull area. No râles. There is a small area of blowing breathing and bronchophony behind and a few râles below the dull area. About 15 c.c. of bloody fluid withdrawn from the third space anteriorly. No acid-fast bacilli in sputum. Died suddenly October 5th.

x-Ray shows a dense homogeneous shadow occupying the upper two-thirds of left chest, with displacement of upper portion of mediastinum to the right. There is no cardiac displacement. There is paralysis of left vocal cord. Patient practically afebrile. Highest temperature 99.6° F.

Postmortem Findings.—In upper lobe of left lung there is a grayish-yellow new growth (sarcoma) extending into the superior mediastinum. The tumor infiltrates pericardium and arch of aorta. The left recurrent laryngeal nerve is embedded in tumor tissue. There is a healed tuberculous lesion in the left apex.

Case X.—A. C., male, aged eight. Admitted September 26, 1923. Died October 26, 1923.

Complaints.—Pain in left side of chest. Cough for five weeks.

Personal History.—Pneumonia and dry pleurisy at three years of age. Whooping-cough at age of two.

Family History.—Father, mother, and 4 brothers and sisters, all alive and well.

Present Illness.—Five weeks ago child was playing with younger brother, who hit him on the left arm with his fist. Patient immediately complained of great pain on left side of chest, cried, felt very faint, and turned pale. Since then he has had a cough paroxysmal in character and chiefly at night. The cough causes great pain in left side of chest. There is no expectoration. He has been feverish since onset of illness, temperature reaching 101° to 102° F. at times.

Present Condition.—Is well nourished and well developed. Glands are negative.

Resp. Sys.—Chest is well formed. The whole of left side of chest is prominent, the intercostal spaces are obliterated. Fremitis is absent on left side. The left chest is dull, the dulness extends 1 inch to right of sternum. Breath sounds and vocal resonance absent on left side. The heart is displaced to the right. Aspirated several times; 10 c.c. of blood-stained fluid obtained. More could not be obtained at one time.

x-Ray shows the presence of a dense homogeneous shadow occupying the entire left chest, with obliteration of the left half of diaphragm. The heart and trachea are markedly displaced to the right.

October 18th: Rib resected and some tissue scraped out. Report of sections shows masses of small round-cells with numerous mitotic figures.

Postmortem Findings.—The left pleural cavity is completely filled by a grayish, soft, mushy tumor growth. This has completely replaced the lung tissue. The mediastinum and heart are displaced to the right of the vertebral column, and the right lung is compressed to half its normal size. There is an old fracture of the left second rib, with complete union. No evidence of tumor of the rib. No tumor elsewhere.

Diagnosis: Rapidly growing round-celled sarcoma.

Case XI.—L. D., male, aged forty-eight. Admitted April 27, 1920. Died April 27, 1920.

Complaints.—Shortness of breath. Cough. Bloody expectoration.

Personal History.—Negative. Used alcohol to excess.

Family History.—Negative.

Present Illness.—For past two years has had dyspnea, often having to sit up in bed. He worked until five months ago. At about this time pain developed in the right side. This has been more or less constant and at times severe. Since then he has had palpitation of the heart and frequent perspirations. His expectoration has been blood-tinged for a year.

Present Condition.—Is very fat. Face, lips, and hands cyanosed. Dyspnea marked, expectoration bloody. There is dulness of the whole of the right side with absent breath sounds. There are many crackles over the whole area. There are no acid-fast bacilli in the sputum. Death occurred soon after admission.

Postmortem Findings.—Sarcoma of right lung—massive. Three lobes. Metastases left lung, upper and lower lobes. Invasion of pleura by direct extension, right. Perforation of pericardium right side by invading tumor mass.

Hemopericardium.

Case XII.—A. M., male, aged forty-five. Admitted May 13, 1923. Died August 16, 1923.

Complaints.—Pain in the left chest. Cough and hoarseness of voice. General weakness. Night-sweats.

Personal History.—Negative. Uses alcohol and tobacco in moderation. Was overseas for three years and was discharged as A1 in 1919.

Family History.—Mother died of cancer of the breast, age unknown.

Present Illness.—After discharge from the army he worked until June, 1922, when he complained of fatigue and began to lose appetite. On August 5th while bowling he had a chill and complained of pain in his left side. This was immediately followed by two weeks of high fever and drenching sweats. In the

latter part of September he was able to get up for a little while. During this time he coughed a great deal, with scanty expectoration, which on one occasion was blood-tinged. In November an encysted empyema was drained. Following this his condition improved for about six weeks. The cough, however, persisted and he spat up a considerable quantity of blood and pus.

Present Condition.—Is much emaciated. Over the left side in front is a flat note from apex to base and behind from the seventh rib to the base. In the upper portion of the dull area there is blowing breathing. Otherwise breath sounds are diminished. On May 27, 1923 aspirated in the fourth space of left axilla. Needle met solid tissue in all directions except upward, from which region blood was withdrawn.

x-Ray examination shows the presence of a fairly dense shadow occupying the upper half of the left lung field, with several circular areas of increased radiability scattered throughout. Heart and mediastinum are displaced to the right.

The Wassermann negative.

There is marked paresis of the left vocal cord.

The illness was febrile throughout.

Postmortem Findings.—Extensive sarcoma of the left lung and the mediastinum.

Case XIII.—J. S., male, aged fifty. Admitted November 12, 1920. Died November 13, 1920.

Complaint.—Dyspnea.

Personal History.—Negative. Very heavy drinker. No history of syphilis.

Family History.—Negative.

Present Illness.—In January, 1920 he had heavy cold on chest, which has been present ever since. Pain across lower right chest. Darting in character, dull ache below. Cough with considerable expectoration. There is some difficulty in getting food down. It appears to stick in the chest.

Present Condition.—He is very thin. He is in acute respiratory distress. Examination of chest is difficult, as respirations are so noisy and the man is so ill.

x-Ray shows heart shadow and mediastinum displaced to the right, outline of right diaphragm not being made out. Lower portion of right chest occupied by a homogeneous grayish shadow confluent with enlarged and blurred hilus shadow. The left lung in the middle third of the subclavicular region is mottled, the same condition being present to a lesser extent at the left base.

The patient died suddenly the day after admission.

Postmortem findings:

(1) Sarcoma of mediastinum; metastasis, right lung, pleura, ribs, liver, stomach, and mesenteric glands (few).
(2) Lobar pneumonia of right upper lobe.
(3) Pleuritis—old fibrous right side.
(4) Emphysema (compensatory) of left lung.
(5) Adhesions, fibrous—between gall-bladder, duodenum, transverse colon, and distal portion of ascending colon.
(6) Hypertrophy of prostate, moderate.

Conclusions.—1. Sarcoma of the mediastinum and lungs is a relatively common disease. In addition to the 13 cases above reported, 6 others were diagnosed as such clinically, and 1 died in the ambulance on the way to the hospital, which postmortem showed to be another.

This covers a period of four years, 1920 to 1923. There were 6208 admissions to the Medical Wards during that period.

2. It is often difficult to say which is involved first, the mediastinum or the lung, unless the case is seen early.

3. The mediastinum is more often the original seat of the growth.

4. Sarcomas grow slowly but extensively, and select the line of least resistance, but some definitely invade such structures as the pericardium, heart, etc.

5. They may metastasize in the lung, and if a vein is ruptured metastases may be found in any organ.

6. There may be extensive involvement for a long time with only a moderate loss of weight, and the other signs which one usually associates with malignant disease.

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CLINIC OF DR. A. MACKENZIE FORBES

CHILDREN'S MEMORIAL HOSPITAL

THE EARLY TREATMENT OF POLIOMYELITIS

THIS patient, K. H., aged nine, is brought to your notice because he is suffering from a common form of paralysis, which is not only seen in our big cities, but is endemic throughout the country at large.

On September 7, 1920 the patient complained of pain in his back. This pain lasted for a week, during which time he was kept in bed. The practitioner who examined him concluded "that he was suffering from rheumatism," but as he was somewhat puzzled, on the fifth day he called in a consultant, who suggested that the child might be suffering from infantile paralysis. On the ninth day I was called in to confirm the diagnosis made by the former consultant.

The boy was suffering from a very definite paralysis of the muscles of the spine. The anterior abdominal muscles were so weak that the abdomen was ballooned. The right deltoid was weak, and both lower extremities were affected with a paralysis which seemed to have no regular distribution, but rather affected certain muscles in one lower and other muscles in the other lower.

The boy was a pitiable cripple, and although there was a cessation of pain after the first week, he was unable to sit up and, indeed, it was impossible for him to turn himself in bed.

It has always been my custom, in teaching students, to impress upon them the necessity of deciding whether a patient is suffering from a paralysis or a pseudoparalysis. Again, we should make sure whether the patient is suffering from a lesion of the upper motor neuron or the lower motor neuron.

Of course, it would seem hardly necessary to impress upon medical men the importance of being able to differentiate between a paralysis and a pseudoparalysis, yet it has been our experience that infants suffering from such lesions as scurvy are sometimes considered to be the victims of poliomyelitis. Again, a child with an inflammatory condition about a joint may be said to be suffering from a paralysis, whereas the child is not paralyzed at all, but rather the muscles are in spasm and the joint is held in immobility through nature's endeavor to protect.

The importance of discovering whether our patient is suffering from a lesion of the upper or the lower motor neuron is based on the fact that we cannot make a rational diagnosis unless we are able to give the approximate position of the lesion.

The upper motor neuron, of course, extends from the motor cortex to the lower extremity of the pyramidal fibers, whereas the lower motor neuron is that part of the nervous system extending from the anterior cornual cells to the muscle-fibers.

To differentiate between the lesions of these two parts we must appreciate that the lesions of one produce symptoms entirely different from the lesions in the other. In the lesions of the upper motor neuron there is a motor paralysis, there is spasticity without wasting, the electric reactions are entirely normal, and there are increased reflexes, and probably pathologic reflexes.

In a lesion of the lower motor neuron there is a motor paralysis combined with flaccidity and muscular atrophy. The reaction of degeneration is present, and there are decreased reflexes.

It is thus often easy to differentiate between lesions of the upper motor neuron and lesions of the lower motor neuron.

The patient before us is suffering from a very definite flaccid paralysis. This flaccid paralysis has affected the arms, the trunk, and the lower extremities. There is muscular atrophy, and if we were to take the electric reactions we would find that the reaction of degeneration is present. Let us examine the

reflexes. There are no pathologic reflexes, and the normal reflexes are either absent or much decreased.

Having gained this information, we appreciate that we are dealing with a child who is suffering from a lesion of the lower motor neuron. Now what is the most common form of paralysis due to a lesion of the lower motor neuron? In my experience poliomyelitis is by far the most frequent cause of such a paralysis. Can, then, our patient be suffering from the lesions of this disease?

Let us study his history. The onset was sudden; the practitioner who first was called in to make an examination could not be certain whether the boy's temperature and indisposition was due to rheumatism or some other yet undiagnosable indisposition. To his horror, he noticed, as the boy began to improve, that there was very definite weakness developing in the boy's shoulder, lower extremities, and back. Indeed, the boy could not sit up. Consultants were called in. The boy was undoubtedly suffering from an attack of poliomyelitis.

Let us consider this condition, which, as I have said, is quite frequently seen in both the cities and hamlets of Canada and the States. It is a communicable, infectious disease which is seen both epidemically and sporadically.

The most characteristic lesions are in the anterior horns of the spinal cord, although any part of the central nervous system may be affected. I have already inferred that it is the most common form of flaccid paralysis found in children. I may go further; it is the most common form of any kind of paralysis seen in children.

About 80 per cent. of cases in children are seen during the first four years of life, but no age is exempt from this disease. I remember a student at McGill—one of our own medical students—being affected. In his case a bulbar paralysis resulted. He died.

It is interesting to note that the epidemics of this disease almost invariably occur in the warmer months of the year. Indeed, in my experience, even the sporadic cases are more liable to be found in these months.

The disease is evidently contagious or, perhaps I should say, communicable. In my practice I have seen several families where 2 children have been affected at the same time. I do not know whether some families have a greater predisposition than others; I know that in one of my families, where the child was affected with this condition during his first year of life, thirteen years later his brother, who was three years old, was affected with the same disease. Of course, this might have been a coincidence.

I do not think that there is any doubt that healthy people may act as carriers; indeed, I would say that this has been proved. For instance, there may be a case of poliomyelitis in a village far distant from another village. If one of those who have been in contact with the patient in the first village happens to visit the distant village, it is not improbable that his visit will be followed by one or two cases in the new village.

It has been noticed that the incubation seems to be within nine or ten days; for instance, the second case in a family usually follows the first within ten days.

For some decades we have known that the pathologic changes following the invasion of poliomyelitis are most marked about the blood-vessels surrounding the anterior cornual cells. It was known that we were apt to get a cellular infiltration of small round-cells about and in the wall of the blood-vessels. It was known that this infiltration was followed by degenerative changes in the nerve-cells. It is only within recent years, however, that the organism which caused this infiltration was isolated. This is a filtrable virus which is said to remain in the spinal cord and lymph-nodes for months after an attack. On the other hand, it is probably to be found only in the cerebrospinal fluid in the very early stages of the disease. Perhaps it has already disappeared at the time that the paralysis is noted. Nevertheless, it seems to persist, or it may persist, in the nasal mucous membrane for some time after an attack. Indeed, many think that the nasal mucous membrane is the portal of entry of the virus of poliomyelitis and certainly it is in the nasal secretions that it is carried, or that it will probably be found in carriers.

I have always taught that it is most important to remember that while the permanent paralyses which follow poliomyelitis are due to degeneration and actual destruction of ganglion cells, there are pseudoparalyses or temporary paralyses which are due to obstruction to the circulation or to the toxins eliminated from the virus. The fact that we have temporary paralyses is of the utmost importance. It alters the prognosis. Was it not Osler who said that when we were called in to see a child suffering from this disease, we could give a guarded, though favorable, prognosis. In other words, when a child is first affected many more muscles and parts will be incapacitated than will be found at a later date. The muscles or parts of muscles which are governed by cells which have been destroyed are permanently paralyzed, but the muscles which are governed by cells which have suffered toxemia or pressure will gradually recover. How long may we expect recovery to take place? I teach that this occurs for at least two years. I have called the first two years succeeding an attack of poliomyelitis "the years of degeneration and regeneration," because during this period there is degeneration of muscle which has lost its nerve supply, degeneration due to the stretching of unopposed muscles, and at the same time degeneration due to stretching by gravity. Coincidently, we see the regeneration of the muscles which have suffered a temporary or pseudoparalysis from circulatory or toxic changes about the anterior cornual cells.

There are various forms of this disease. We have cerebral cases, cases where the lesion is almost entirely in the spinal cord. We have bulbospinal cases; we have cases where the meninges seem to be particularly affected, which so simulate the various meningitides that it is hard to diagnose them from meningitis or, indeed, from encephalitis. Besides these, a polyneuritic type has been described, where there is much complaint of pain, but in my experience it is rare that the motor cells alone are affected in any form of this condition. Indeed, it is common to have pain due to an involvement of the posterior nerve roots as well as the anterior cornual cells. Tenderness may persist for months. Of course, in the type of case

where pain is a dominant feature it is important that we should remember to differentiate the condition from a non specific polyneuritis.

One of the important characteristics of poliomyelitis is that there is no order of attack. A muscle here or a muscle there, a part of one leg, and a muscle in an arm. Perhaps the abdominal muscles may be affected. Less frequently the thoracic or respiratory muscles are affected.

Let us observe the patient who is the subject of this clinic. The invading organism has long since spent its strength.

It is of treatment that we should speak, because we feel that in the past our knowledge of treatment has been very vague —may we not say that it is still very vague.

The child is attacked; we may be fortunate enough to make a diagnosis early. While the various sera suggested might possibly have been used for prophylactic treatment, it is doubtful whether they would be of any benefit after the child is attacked, and certainly of little or no benefit after the paralyses have once developed, because, as we have already said, it is most unlikely that at this time the virus still retains its activity. We can, however, make our patient comfortable; treat him, indeed, by rest, and rest is really the best treatment for all such inflammatory conditions.

I have been asked how soon it is wise to begin treatment by massage or electricity. Certainly not at first and certainly not for some time. Legg, of Boston, recently said that orthopedic treatment should be begun as soon as the acute cerebral or febrile symptoms have subsided. I agree with this, if he means that the patient should be placed under the supervision of a surgeon who takes an interest in lesions which cause deformities, but I am utterly opposed to active treatment in the early months following the invasion of this virus. *Rest* should be our form of treatment. I advise rest in recumbency if there has been any extensive involvement of muscles or parts. Besides this, rest to the parts affected is of the utmost importance.

Legg said that the sensitive stage is an important one, and its disappearance is the guide for the beginning of orthopedic

treatment. He said that a duration of six months for this stage is not at all unusual, and that no active treatment should be carried out while pain is present. Undoubtedly, the best treatment for the sensitive stage is absolute rest, hot baths and hot blankets, or baking by electric lights.

I think it is wisest, under our present lack of definite knowledge, to make it a rule that active treatment by massage, electricity, or manipulations should not be undertaken for six months from the onset of the paralyses. This is an arbitrary rule, but we know that in many cases where there is involvement of the posterior roots sensitiveness remains for six months. Surely this outward manifestation of a disordered sensory nervous system points to the fact that the same period of time may elapse before a disordered motor system resumes its equilibrium.

An Australian, MacKenzie,[1] says if rest is the basic treatment of inflammation—and toward this we must suppose that Nature is always striving—it is clear that the cornual cell can be best rested through the muscle. With constant irritation of the muscle, by faulty position, massage, and electricity the utmost is being done to prevent recovery of the inflamed sensory nervous system. It is a fallacy that we must "do something" to the muscle to maintain its nutrition. If a motor nerve be injured, the injury has also affected the muscle, and how can we expect recovery of the muscle after repair of the nerve if we fail to rest the muscle? Not only is muscle rest the most effective agent for repair of injury or inflammation of either muscle itself or of the nervous system with which it is in communication, but the position of rest is the physiologic basis for the re-education of muscle function.

What constitutes muscular rest? is the question of this Australian writer. He replies, A normal muscle cell is possessed of these two properties—the power of contraction and the power of relaxation. The stimulus which causes one set of muscles to contract, causes those which act in an opposite direction to relax. Contraction of the muscle, or elongation of its relaxed opponent, may be described as an irritable state.

[1] William Colin MacKenzie, The Action of Muscles, 1919.

The intermediate position, which is that of rest or equilibrium, is not a state of communication, but is an active state in which the opponents are evenly balanced. The position of rest is the normal position of equilibrium between two muscles or groups of muscles.

If a muscle be weakened or paralyzed, whether from injury to muscle, nerve, or central cell, it is rested when its opponent is in a state of relaxation and elongation beyond the state normally regarded as necessary to produce a condition of equilibrium with its opponent. In this way we prevent a weakened muscle being stretched and irritated by the contraction of its opponent, and we assume that the application of electricity or massage is not allowed to interfere with recovery.

If we leave a paralyzed deltoid unsupported, with the hand hanging at the side, not only have we not true muscular rest, but have, in addition, the weight of the upper limb dragging on a weakened muscle.

So much for the creed pronounced by the Australian. I have already stated my belief. A patient who has suffered from poliomyelitis should be treated by rest and protection for six months. It is only after six months that I endeavor to re-educate muscles and groups of muscles by volitional exercises, by massage, and even by electricity. It is only then, in my opinion, although I speak very arbitrarily, that we can have reasonable assurance that the active result of the inflammatory condition has ceased to obtain in the nervous system, and as I look upon the nervous system and the muscular system as being so intimately connected that they are as one, I prefer to treat by rest, both local and general, until this arbitrary period has passed.

After the termination of the six months of rest volitional muscular training should be begun. It is important to appreciate that volitional muscular treatment must be begun from the "zero" position of rest; in others words, as Mackenzie says, when we ask a patient with a deltoid paralyzed from poliomyelitis to commence work, while sitting up with the upper limb hanging at the side, we are asking damaged cells to begin

work at the greatest disadvantage—asking a baby to walk before he has learned to stand. We must ask muscles to begin work at "zero," to commence work by beginning with a minimum function, or at a point where their load is at a minimum. Though we speak of a muscle anatomically as being a flexor or extensor, we really refer to the maximum function of a muscle. The question arises as to the recognition of the minimum of muscular function, for it is only by the recognition of the minimum that the muscle may be ultimately coaxed up to the maximum.

We begin at "zero," and although we may think that the amount of work at this minimum is slight, we must remember that it really represents the maximum function of the muscle for the time being, and as such may soon become exhausted. It is most important that we realize that every group of muscles has its opposing group; indeed, there is such a thing as muscular balance, and if this balance be interfered with by a paralysis or a weakness of a muscle or a group of muscles, the unaffected muscles are unopposed and tend to become contracted and thus cause deformities. For this reason it is of the utmost importance that, when we treat our patient by rest, this rest shall be both local and general. What is local rest? That posture which prevents the stretching of one group of muscles and the contraction of another group of muscles. This posture must be maintained by brace and bandage during the time when contractures may occur. It is due to lack of appreciation of this fact that deformities occur. Muscles are allowed to contract by lack of opposition. Muscles which suffer paralyses or pareses are allowed to become stretched, and through this very stretching the little function which remains in them is gradually destroyed. These deformities from contraction and stretching are seen frequently at the knee-joint, when, for instance, the quadriceps is weakened by disease or injury, the hamstrings may contract or become shortened through lack of opposition and through the effects of gravity. The same thing obtains when we have paralysis of the deltoid. The pectoralis major tends to become contracted. At the ankle, if the dorsiflexors are par-

alyzed, we get an equinus deformity due to contraction of the plantar flexors or extensors. In all of these deformities gravity, of course, plays a prominent part. How much care is indicated in the treatment of paralyzed muscles! In the case of a paralyzed deltoid the dragging weight of the upper limb—operating whether the patient is erect or lying down—permits of contraction of the unopposed muscles. All these deformities can be prevented. Rest and posture to the parts affected are not only indicated, but will save the patient from further loss of power. The treatment of all these conditions, however, must be based not only on physiologic but anatomic principles. It is useless to hope to control a muscle by applying a splint without regard to the attachments of the muscle to be controlled.

When a patient like the boy whom we are presently studying is presented for examination, the muscles which we suspect to be affected must be tested. It has been the custom in the past to test these with electricity, but I am inclined to agree with our Australian friend, who says that there is only one true test for muscular action—the volitional test scientifically applied. This is the only test which takes into account the two elements of muscular action, the contraction of one muscle and the relaxation of its opponent. By the volitional test we are able to determine the minimum action from which the muscle can be re-educated up to the maximum. In making these tests we must always consider the effect of gravity; the placing of the origin and insertion of the muscle as nearly as possible on a level is most important, as only when we do this can we discover those minimum powers of contraction which remain in muscles which otherwise might be considered irremediably destroyed.

We believe that graduated re-education is not a source of irritation to a recovering nerve as long as it is not undertaken too early. I am inclined to think that the Australian begins his muscular re-education very much earlier than is done in our clinics, where we have felt that in many cases of poliomyelitis there is irritation about the nerve-centers for a period often approximating six months.

The treatment prescribed for the patient who is the subject of this clinic was *rest* both by recumbency and *rest* to the parts affected by posture and splint. Later he was treated by massage and volitional training.

Let us examine him. He suffers no fixed deformity. It is true that spinal support in the form of a light brace is necessary to maintain the spine in a normal position. It is true that certain of the abdominal muscles and the muscles of the extremities are much atrophied and weakened. These will require volitional exercises during his age of growth. There are no contractures to be relieved. No tenotomies are necessary. There are no indications for operative procedures.

It is true that in many such patients muscle-grafting is necessary to secure muscle balance and assure function. It is true that operations on bones and joints are often indicated to secure stability and to relieve deformities, but in this patient, happily, support and support alone is all that is at present indicated.

CLINIC OF DR. A. ARMOUR ROBERTSON

CHILDREN'S MEMORIAL HOSPITAL

SPINA BIFIDA OCCULTA WITH SPINAL CORD LESION

THE patient whom I wish to present is a boy thirteen years of age who, having been seen previously in the Outdoor Department of the Childrens' Memorial Hospital, Montreal, was admitted to the wards on June 11, 1923 for further observation.

He complains of great difficulty in walking owing to weakness and spasm in the legs.

The following history has been obtained: His birth was normal, no instruments being used. He was breast fed for nine months and was quite healthy as an infant and child. He began to walk at one year old and to talk at about eighteen months. He began to attend school at six years old, and has always been bright and intelligent. There is no history of any motor or sensory disability at this time, and it is stated that he was able to run and play with the other boys. In fact, careful questioning of the parents failed to elicit any evidence that he was not in every way a normal child.

There was nothing of moment, bearing on the condition, found in the family history. Both his father and mother are alive and in good health, and he is one of 6 children, the others all being healthy.

In February, 1921 he had a toboggan accident. It was not thought much of at the time, and he was not confined to bed, nor did he suffer much, if any, pain immediately after the accident. However, in about three months he began to limp a little, and some pain was complained of in the region of the left hip. This condition of weakness increased gradually, and it was noticed that both legs were involved. The onset was gradual, so

that at first he only felt weak after standing for about ten minutes. This weakness was localized by him about the knees. Pain was not a prominent feature.

In August, 1921 he was taken to another hospital, and though the radiograms taken did not show any definite lesion, the condition was diagnosed as due to tuberculosis of the left hip and the hip was put up in plaster for three months. There was no improvement after the removal of the plaster. About the end of 1921 it was noticed that there was tremor in the legs. He was taken back to the same hospital in September, 1922, when it was realized that the disability was due to some spinal cord trouble.

When he came under observation in this hospital it was seen that he was a fair-haired rather slim boy thirteen years of age. His color was moderately good. He was bright, with intelligence certainly normal, if not above normal. He was not suffering any pain and did not complain of anything except the disability due to the leg condition.

On examination no abnormality was found in the respiratory or circulatory systems. His teeth were in rather poor condition and his tonsils were enlarged. No other abnormality was found in the digestive system. His eyes were clear and there was no nystagmus or strabismus. His pupils were equal and reacted to light and accommodation.

On examination of the back it is noticed that there is a scoliosis with curvature to the right, and showing some rotation of the vertebræ. There is a large vascular nevus in the lumbar region, extending more to the left than to the right, and crossing the spine from the second to the fourth lumbar vertebræ (Fig. 316). On palpating the spine in the vicinity of this nevus there is a feeling as if the spines cannot be felt as well as they should.

On examination of the locomotor system marked abnormalities are found. He is only able to walk with great difficulty with the aid of a cane and the gait is markedly spastic. The knees are kept partly flexed and he has difficulty in raising the feet from the ground. So far as could be tested there is no ataxia.

There is marked loss of power in both legs, but more so in the left leg, in fact, some movements cannot be carried out voluntarily. Thus, on the right side the toes and ankles can be voluntarily moved in all directions, though eversion of the ankle is weak, but the right foot is held inverted and cannot be voluntarily everted, nor can the toes be moved. There is a fair degree of voluntary extension and flexion of the knees on both sides. All movements, however, are carried out slowly and

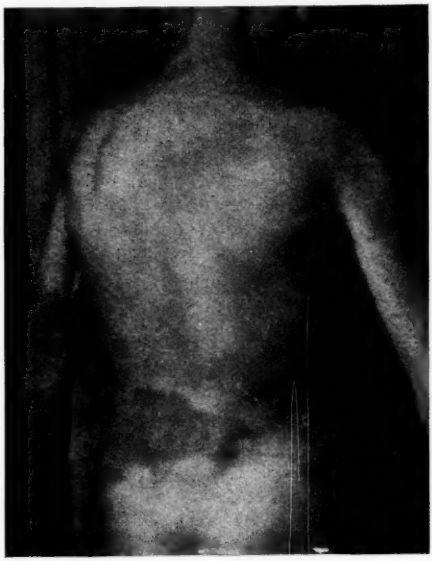

Fig. 316.—Photograph of back showing nevus.

with difficulty on account of the general hypertonic condition of the muscles, and, in addition, almost any attempt at movement causes a very marked coarse clonic spasm of the whole limb, which the patient attempts to control by pressure with the hands on the knees. This clonic spasm is more marked in the right leg, which is otherwise in better motor condition.

Measurements of the legs and thighs show that there is relative atrophy of the muscles of the left leg to a slight degree.

Electric examination showed faradic response present in all muscles of both legs, but this required a stronger current in the peronei, more markedly so in the left leg.

The epigastric reflexes are present, but the lower abdominal and cremasteric reflexes cannot be elicited. In the legs all the reflexes are exaggerated, thus the knee- and ankle-jerks are much increased, and rectus and ankle-clonus are very easily obtained. Babinski reflex is very positive on both sides.

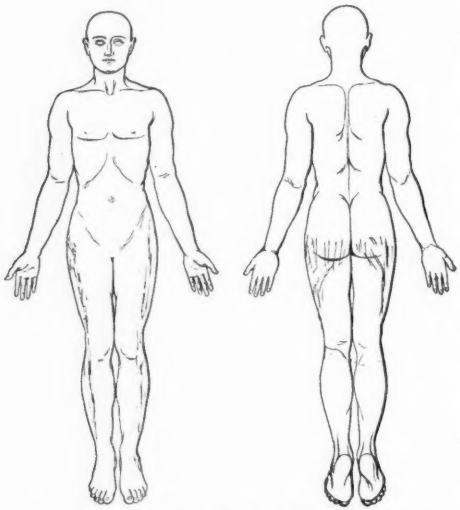

Fig. 317.—Area of diminished sensation to pain.

Examination of sensation shows definite and wide-spread dissociated anesthesia.

Sensation to touch (cotton wool) is well felt and quickly and accurately localized all over both legs, though there is a slight relative loss on the outer aspect of the left thigh.

Sensation to pin prick is well felt over part of the legs, but is not so well felt, as such, over certain parts. Of course, on account of ordinary sensation being present, he can feel the pin prick as touch, but over the abductors of both thighs and ex-

tending round the back of the thighs over the glutei and also over the lower part of the legs, especially on the outer aspect, there is definite loss of pain sensation. This loss seems to be somewhat greater on the right side than the left (Fig. 317).

Sensation to heat and cold shows more wide-spread loss. He is being tested by means of test-tubes filled with water with a wide variation in temperature, thus not requiring fine appreciation. He finds these hard or impossible to distinguish over the

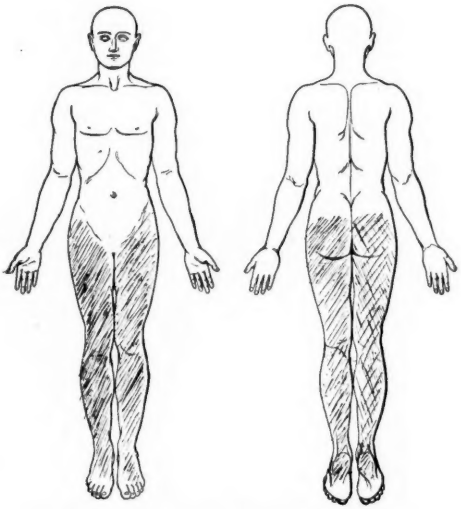

Fig. 318.—Areas of thermo-anesthesia.

surface of both thighs and legs up to the line of the groin in front, and to a line running across the gluteal region behind (Fig. 318). He frequently guesses wrong, and volunteers the information that he finds it difficult to distinguish, and that his answers are largely guesswork. Careful examination of the body above this area shows that he can quickly and accurately distinguish heat from cold and that other sensory powers are also normal.

The motor power and co-ordination of the upper part of the

body and head and upper extremities show no abnormality with the exception of the scoliosis previously mentioned. The reflexes in this part are normal.

Fig. 319.—Skiagram showing spina bifida at eighteenth sacral.

There is no incontinence of urine. The only difficulty that he complains of being that at times there is some difficulty in starting the stream.

The cerebrospinal fluid showed 30 lymphocytes per cubic

millimeter and globulin tests were positive. Wassermann reaction was not positive.

Diagnosis.—We have here a patient who shows a marked condition of spastic paraparesis with dissociated anesthesia in the lower extremities, and the history tells us that the condition has come on somewhat gradually in rather less than three years, with progress apparently more rapid at first than at present. The patient was apparently functionally normal before this time, though examination of the back shows the presence of a large nevus, and it is a matter of common knowledge that such conditions are frequently associated with spina bifida occulta, and, indeed, in this case a radiogram (Fig. 319) shows this condition to be present in the first sacral vertebra, the commonest site.

There is also the interesting fact that the symptoms all manifested themselves a short time after the boy had received an accident.

The condition of dissociated anesthesia is made possible owing to the fact that there are two paths by which sensation travels up the cord, so that some fibers ascend in the posterior tracts of the same side, decussating in the medulla, while others pass over, shortly after entering the cord, by the commissure and go up in the lateral tract of the other side. Tactile sensation is conveyed by both these tracts, while pain and heat are carried by the latter. Thus a lesion in the region of the central canal will interfere with conduction through the commissure and prevent the perception of sensations of heat and cold in the part affected, while tactile sensation may remain relatively, if not quite, normal. This condition is commonly associated with syringomyelia, as in this condition cavity formation, secondary to gliosis, is found in the region of the central canal of the spinal cord, and this condition might be first suggested as an explanation of the conditions found in this case.

However, this case shows some marked differences from the ordinary history of a case of syringomyelia in that the gliosis and the secondary cavities are usually widely extended longitudinally in the spinal cord, and the first symptoms are nearly

always in the upper extremity. Though paraplegia may manifest itself, it is nearly always a late symptom, and is seldom of very great severity. In the majority of cases the symptoms first appear considerably later in life than in this case, though cases have been described beginning in early childhood (Holmes, Nelson's Loose Leaf Med., article "Syringomyelia").

Several different causes have been given for the occurrence of syringomyelia, but it seems undoubted that some congenital anomaly must be at the bottom of many, if not all, of the cases, and when we recall the formation of the spinal cord, this would seem reasonable.

The central canal in early fetal life takes up a large part of the neural tube and becomes gradually smaller by obliteration by ingrowth of neurogliar and nervous elements, and it is reasonable to suppose that in certain cases during this development remnants of fetal or neurogliar tissue may be left. These remnants may remain dormant for a variable time, and then, for some cause, in some cases undiscovered, in others apparently secondary to trauma, they begin to grow, causing a gliosis. Secondary degeneration in this glial tissue caused the formation of cavities which constitute the peculiar pathologic findings in this disease.

The diagnosis of syringomyelia does not seem to be quite satisfactory in this case for the reasons mentioned, that is, on account of the symptoms being confined to the lower extremities and to the rapid appearance and high grade of the spastic paraparesis. It is true that it is stated in all text-book articles on syringomyelia that spina bifida occulta is frequently associated with this condition, and such cases are recorded (Kleppel and Teil, Presse Med., December, 1921), but I would suggest that in some at least of these cases the spina bifida was the primary lesion, and the cord disturbances were secondary to this. This point will be taken up later.

The dissociated anesthesia, associated with the spastic paraparesis, serve to distinguish this case from other spinal affections found in childhood such as acute anterior poliomye-

litis, Freidreich's ataxia, progressive muscular atrophy in its various forms.

Otto Beck in an exhaustive article (Ergebnisse der Chirurgie und Orthopädie, 15th Band, 1922) has reviewed the various deformities and other disabilities associated with spina bifida occulta, and to this article I wish to express my indebtedness.

Spina bifida occulta is a condition which may be recognized in some cases by palpation, but in many can only be recognized with certainty by means of radiograms. There is to a variable degree non-union in one or more spinous processes. The spinous process or processes may be completely absent, or they may develop in part, but without union of the two sides. This is so in this case, where the radiogram shows that there is a space between the projections from the two sides with apparently some overlapping.

This anomaly may occur in various parts of the spinal column, but most frequently in the lumbosacral region, as this is the last part of the spine in which union normally takes place. Many cases show no abnormality in the skin over the lesion, but in a considerable proportion there is a growth of hair usually of the same color as the hair of the head. In other cases other skin changes have been described, and among these are nevi, such as that present here.

In some cases there may be no symptoms, and the condition may be an accidental find, but in others more or less serious deformities and paralyses may develop or be present from birth. Among the deformities ascribed to this condition are many cases of club-foot and claw-foot. The condition differs from cystic spina bifida in the absence of visible cystic growth, though intermediate forms are seen in which there is a small hard nodule visible. The difference is probably due to the fact that, in occulta, intra-uterine healing has taken place of a cystic spina bifida. In many cases this healing is not complete, but there is left a small mass of varying size and of myo-fibro-lipomatous structure. This may be palpable subcutaneously, or may be deep enough to evade palpation. Just as in cystic spina bifida the cyst may have different coverings according to the depth

in the cord from which it springs, so these masses may invade the cord or be superficial to it. They are one cause of the pathologic signs and symptoms by their pressure on the cord, and from the fact that nerves issuing from the cord are frequently involved in them.

Another cause of pathologic signs is dependent on the fact that in early fetal life the body segmentation is more simple and that the cord extends along the whole length of the spinal canal, and so the nerves issue at right angles to the cord. With growth of the fetus the cord does not grow in length with the spine, so that the cord "ascends" in the spinal canal and the cauda equina is formed. In spina bifida occulta, owing to adhesions, this ascension of the cord is hindered in varying degrees, and the cord may reach to an abnormally low level in the spinal canal. In addition, bands of adhesion are formed which by the growth of the spine are stretched. These are another factor in causing abnormalities by pressing on the cord and so hindering its proper functioning. Owing to the increased and frequently rapid growth near puberty, this pressure and traction is at that time often increased, and symptoms appear, or, if present, are apt to be aggravated.

In this case shown the symptoms first appeared at a time when growth was rapid, and consequently this may have been the cause rather than the trauma reported.

This diagnosis of spina bifida occulta with secondary cord lesions seems to be the one which best fits the symptoms, though the extent of the dissociated anesthesia is greater than in most cases reported, as it involves on both sides everything below the level of the first lumbar segment of the cord. This symptom of dissociated anesthesia Beck reports to have been present in many cases. It might be ascribed, if congenital anomalies are not present in the cord, to interference with the circulation of the cerebrospinal fluid and the causation of a condition of hydromyelia.

The hypertonic paresis of the muscles present implies that there is interference with motor conduction above the lower lumbar region, as the lesion is of upper motor neuron type in

these muscles, whose cord supply comes from at least as high as the fourth lumbar segment. The more marked paralysis in some muscles, as the peronei, especially on the left side, in addition to the diminution in all sensation in the same area, would seem to indicate that there may, in addition to the cord involvement, be involvement of some nerves in the cauda equina.

The diagnosis in this case is not, as it is, perhaps, in too many cases, simply undertaken as an attempt to anticipate successfully the subsequent findings of the pathologist, but has an important bearing on the treatment. If the case were one of syringomyelia, the only therapeutic measure for which any claim has been advanced as being able even to arrest the course of the disease is treatment by means of the *x*-rays. Cases have been reported of late years, especially in France, where this treatment has apparently given good results (Coyen, L'Hermitte, and Beaugard, Bull et Mem. de la Soc. des Hôp. de Paris, 3 Mars, 1922, and L'Hermitte, Paris Med., October 1921). This is with the exception of some comparatively rare cases where syphilis has apparently been the underlying factor and where good results have been reported from active antisyphilitic treatment (Oppenheim).

With regard to spina bifida occulta the case is somewhat different. Surgical interference for the relief of the underlying condition has been undertaken by various surgeons with varying degrees of success. This operation consists in opening up the spinal canal and removing the myo-fibro-lipomatous masses, if present, and also freeing the adhesions. Among these surgeons was Sir Robert Jones (Brit. Med. Jour., 1891).

Beck, in the article mentioned above, cites Cramer and also Ludloff as having operated in this way. Cramer, he reports, has had many cases, and has had good results in a good proportion. The operation is not to be undertaken lightly and would be contraindicated if the disability were slight and nonprogressive. For example, in cases where the only manifestation was club-feet there would be no justification for other than local treatment. In cases where the disability is greater and is

not amenable to local treatment, he considers that the more serious operation is justifiable.

In this case no attempt at operation has been undertaken, but it will be seriously considered, as the disability is so marked, and even an improvement, as, for example, a reduction of the spasm, as has been reported in even long-standing cases, would justify the procedure.

CLINIC OF DR. D. GRANT CAMPBELL

MONTREAL GENERAL HOSPITAL

THE DANGER OF PREGNANCY IN HEART DISEASE

GENTLEMEN: Some of you will remember this patient when she was first referred to this hospital (September 26, 1923) from the Out-patient Department of the General Hospital. At that time her examination showed a well-developed woman pregnant about six months, and with symptoms and signs of early cardiac failure as evidenced by palpitation, breathlessness, cough, some cardiac hypertrophy, a presystolic murmur at the apex, a rapid, soft pulse, and moist crepitations at the bases of both lungs.

This was her seventh pregnancy and she gave a past history of rheumatic infection and of influenzal pneumonia. You will recall that she was put to bed at once, and though under appropriate therapy, she improved but very slightly, her breathlessness and rapid pulse remaining as on admission. In consequence, after one week's observation, she was advised that a termination of her pregnancy was considered the wisest course. She refused to accept this advice, as did her husband, and she left the hospital the same day.

Six days later she was readmitted almost *in extremis*, orthopneic and cyanosed, with marked edema of the lungs and feet. The pulse was 140 and scarcely palpable, her blood-pressure 98 systolic, 70 diastolic, her heart markedly dilated to right and left, and the heart sounds scarcely audible. Her condition remained very poor for some days, but she gradually improved and was kept in bed for a month. By the time her own condition warranted any interference it was deemed wiser to wait and make sure of a viable child, so she remained in the hospital until she was delivered three weeks ago in a rapid, easy, spon-

taneous labor. For two days postpartum she was in poor condition, but she rapidly improved, and, as you can see for yourselves, is now in fair condition, able to sit up in a chair for a short time daily. You will notice from her chart the effect of the fetus on her pulse—antepartum it remained constantly above normal, mostly 100 or more, but subsequent to delivery it returned to normal.

You may ask, in view of the final outcome, why she was advised to have her pregancy terminated when first seen at six months. In answer I think we may safely say that had she not remained in hospital under treatment she would not have survived, and that it is impossible so to treat the great majority of such cases. Moreover, there is little doubt that her heart muscle has suffered severely, and that her life has been perceptibly shortened by this pregnancy.

Our advice was based on a study of the records of this hospital during the past fifteen years. During this period there were 15,948 admissions, of whom 159 cases were stated to be suffering from associated heart conditions; practically 1 per cent. of the admissions. The reliability of the records from the point of view of cardiac diagnosis is satisfactory. The great majority of the cases prior to 1918 had been seen and examined by one of the senior physicians of the General or Royal Victoria Hospitals, since which date I have had the privilege of watching most of them. That there are some classified in the wrong group is probable, and that there have been other cardiac cases admitted, more especially to the private wards, which did not show any resulting distress, and were, in consequence, not recorded as such, is certain, but the margin of error is not great, and does not, I think, vitiate our conclusions. In addition, a certain number of cases of heart disease which entered the hospital for rest and treatment because of a breakdown in the earlier months did not return for confinement; these are not included, as they do not help to answer the question set before us.

For the purpose of this study the cases have been grouped as follows:

Group 1. Cases showing mitral stenosis.
Group 2. Cases showing mitral regurgitation only.
Group 3. Cases showing aortic regurgitation, with or without mitral regurgitation.
Group 4. Cases showing mitral stenosis and aortic regurgitation.
Group 5. Cases showing auricular fibrillation.
Group 6. Cases showing myocarditis.
Group 7. A group of unusual and rare types of cardiac disease.

1. **Cases Showing Mitral Stenosis.**—This is by far the largest group. There are recorded 67 labors by 57 women. Of the women, 31 were primiparæ, of an average age of twenty-six years; 18 were pregnant for the second time; 1 was pregnant for the third time; 1 was pregnant for the fourth time; 2 were pregnant for the fifth time; 4 were pregnant for the sixth time; 2 were pregnant for the seventh time; 2 were pregnant for the eighth time; 4 were pregnant for the ninth time; 2 were pregnant for the tenth time; 4 patients died in hospital (6 per cent.), 1 before delivery and 3 within a few days postpartum. In addition, 5 are known to have died within (about) twelve months after confinement as a result of heart failure. The children born of these women averaged between 6 and 7 pounds at birth and seemed in every way as healthy as the average child, but 10 of the 69 children involved were sacrificed at the time of labor or died within a few days afterward. Although 2 of the 4 women who died were primiparæ and another was having her second child, it is strikingly evident from a study of the case reports that the danger materially increases with each pregnancy; most of those coming in for confinement in their later pregnancies did so because of evidence of cardiac decompensation during its course; that they did not die was due to the care and judgment shown in the conduct of their cases. Undoubtedly, many of them went out to a life of semi-invalidism; their hearts showed so much damage at the time of confinement that one could hope for no other result.

As a group, cases with mitral stenosis during pregnancy

have considerable distress due to dyspnea and always require careful oversight, for they may at any time show symptoms demanding immediate interference. Decompensation is usually gradual and to be foreseen and relieved, as in our case of today, but how tragically rapid may be its onset is evidenced by the history of 1 of our 4 fatal cases. This patient had an old well-defined stenosis which had never given her any undue amount of distress. She was forty-two years of age and pregnant for the *ninth* time. She was being carefully watched by a thoroughly competent physician, when suddenly, in her thirty-sixth week, she began to suffer from breathlessness and inability to lie down. Her distress increased with alarming rapidity, and thirty hours after the onset of symptoms she was removed to the hospital, where an immediate induction was done, without anesthetic. Within a short time afterward she became very cyanosed and began to cough; respiration became very labored, and both lungs rapidly filled with moist râles. Under oxygen she improved, labor pains began, the packing was removed, and she appeared more comfortable. Four hours later she collapsed again, and in spite of all that could be done she died just forty-eight hours after the onset of symptoms.

That this is not a usual sequence of events is evidenced by the fact that we have had only 1 other case at all parallel. This patient walked one-half mile to the out-door clinic on a cold winter's day when six months' pregnant. There she had a sudden collapse with acute cardiac failure, as evidenced by extreme dyspnea, orthopnea, and cyanosis, and, when examined by me one hour later, considerable edema of the lungs. After eight days' rest in bed she insisted on leaving hospital, and did not return until labor began. She became pulseless after the birth of her child, but finally left the hospital in fair condition. The other 3 cases who died were all brought into the hospital practically moribund, and might, we feel, have been helped had the danger signals been known and recognized.

These signals, authoritatively given by Sir James MacKenzie, are entirely in accord with our findings. He says: "Evidences of heart failure are to be found not in the exam-

ination of the organ, but in the manner in which the circulation is maintained in the different organs of the body. While every organ of the body suffers when heart failure sets in, only a few organs produce recognizable signs. The organs which show the most distinctive signs of heart failure are the respiratory organs and the heart itself. In the case of both these organs heart failure gives rise to distress; in the one case the distress is associated with breathlessness, in the other with pain of a definite kind in a definite locality."

Breathlessness is usually the earliest sign in cases of mitral stenosis. If there are found at the same time persistent crepitations at the bases of the lungs, more especially if there is any dulness on percussion, the heart failure is definitely established.

Treatment.—Close observation throughout pregnancy is essential, particularly for the early signs of cardiac failure. If all goes well beforehand, our records show that these cases stand the first stage of labor well, but that the second stage is that in which the strain is put upon the heart, and that as soon as the cervix is fully dilated, it is good practice to terminate labor by the use of forceps or otherwise, as may be indicated. The danger of an anesthetic does not seem as great here as in other surgical procedures. In nearly every case included in this study a moderate degree of anesthesia was administered —usually chloroform—and in only 1 of the 159 was any alarming symptom attributed to it, and that case rapidly responded to stimulation. Should signs of decompensation occur in the earlier months, there is little likelihood that pregnancy can be allowed to go safely to term; whether it can be allowed to go along sufficiently to obtain a viable child is a question which must be decided in each individual case. One thing is certain—the heart will be permanently damaged.

As to the procedure when interference is needed, opinions differ considerably. Newell urges that all cases, where the patient's condition does not contraindicate it, should have an abdominal cesarean section with sterilization at the same time; if the patient's condition does not warrant this, an attempt should be made to restore compensation and then to empty

the uterus. He urges the use of local analgesia plus morphin and scopolamin for section.

Our experience with such cases at the Maternity Hospital has been limited; only 4 in this group were treated by cesarean section, while 7 were aided by the more simple method of induction of labor. Of the 4 sections, 1 died of cardiac failure forty-eight hours postoperative, leaving a living child; 1 was operated on at the fifth month and sterilized, left the hospital in fair condition, but died in the General Hospital two months later of heart failure; 1 who was well compensated at the time of labor did well and had a living child; the fourth was operated on because of a generally contracted pelvis and had a living child, compensation being at no time upset while in hospital or since.

Of the 7 inductions, 1 died during labor; as already reported 3 were delivered successfully and had healthy children, while 3 were delivered successfully, but lost their babies subsequently while in hospital.

To summarize the treatment of these 67 cases: 26 (or 40 per cent.) had normal labors calling for no assistance and showing no unusual after-effects; 16 were assisted by forceps; 4 underwent cesarean section; 7 had induction of labor; 14 had various surgical procedures which the special circumstances of the case called for. The treatment of the heart condition was mainly by rest in bed, digitalis in the form of the tincture being the drug most commonly used when any further help was considered desirable.

2. **Cases Showing Mitral Regurgitation.**—There are 20 cases in this group. This seems small and out of proportion in over 15,000 cases, but, as a group, these cases give much less cause for anxiety and rarely break down, so that it is likely that a considerable number must have escaped attention, or at least the clinical recording of their disability. The almost invariable summary of the case report is, "Result good, mother and child." Of the 20 cases in this group, 4 were delivered by forceps, the other 16 had normal labors. In none are there any notes or other indications that the heart gave any trouble, except that one was

in hospital for one month's rest antepartum and was the only case (already mentioned) which collapsed under chloroform in the whole series.

3. **Cases Showing Aortic Regurgitation, With or Without Associated Mitral Regurgitation.**—This group furnishes rather unexpected findings. There were so classified 10 patients; only 2 showed any evidence of cardiac embarrassment during pregnancy. One of the 2 collapsed the second day postpartum, developing "acute dilation of the heart." She rapidly improved, however, and went out in good condition. She was next seen eighteen months later, when she returned three months pregnant for a curetage, and at this time her heart lesion was much less evident. Forceps were applied in but 2 cases; all the others were normal cases obstetrically, and only one in addition to the one already described had, while in hospital, a pulse-rate in any way out of the ordinary. Moreover, in contradistinction to the cases of mitral stenosis, those coming in for later pregnancies did not show any proportionate disturbance of the heart.

4. **Cases Showing Aortic Regurgitation and Mitral Stenosis.**—This group is small (7 cases), but sufficiently large to indicate that these cases *always* warrant a grave prognosis. Of the 7 cases reported, only 1 had a normal labor. This one had had some decompensation antepartum, but her labor was so rapid and easy that the baby arrived before she could be removed from the ward to the case room. All the others needed assistance; one was delivered by cesarean section, and all were permanently damaged we consider, as far as their hearts were concerned, by their pregnancy.

5. **Cases Showing Auricular Fibrillation.**—This group, although small, indicates the gravity of pregnancy where fibrillation is present. Most of these cases gave indications that stenosis of the mitral valve had preceded the irregularity. There are 10 cases in this group: 7 pregnant for the first time, 1 for the second, 1 for the fourth, and 1 for the twelfth. Not a single case was normal throughout; 3 had normal rapid labors, but showed heart failure during the puerperium, 5 had forceps de-

liveries, 1 a version and extraction, and 1 died undelivered sixteen hours after admission to hospital, having been brought in with acute edema of the lungs, orthopnea, cyanosis, and thrombosis of the left brachial vein. A second case died on the tenth day postpartum of heart failure; she was admitted in very poor condition and never improved in spite of the liberal use of digitalis. Of the other 8, all but 3 gave indications at the time of discharge, or subsequently, that their hearts were very gravely damaged, and it is very doubtful whether any of them could go through another pregnancy successfully.

6. **Cases of Myocarditis.**—As used here, the term implies cases showing a defective response to effort in whom endocarditis or pericarditis could be excluded as a cause. This group contains 28 cases and it shares with fibrillation the doubtful privilege of being ranked as the most dangerous cardiac complication of pregnancy; 7 of the 28 (25 per cent.) died as a direct result of the pregnancy, while others are known to have succumbed subsequently. Of the deaths, 1, a primipara, entered the hospital with marked heart failure and under the influence of alcohol. Low forceps were applied at the end of the first stage, but the pulse, which was 148 on admission, remained very fast and the patient's heart simply gave out, with death nine hours postpartum. A second alcoholic came in for her fourteenth pregnancy, with signs of decompensation and a constantly rapid pulse (120 +). She had a normal, rapid labor, but had difficulty in the third stage due to a retained placenta. Sudden death occurred on its manual removal; the death, however, appeared to be due to cardiac failure and not to hemorrhage. A third case, a primipara of twenty-eight, was in a moribund condition when referred to the hospital. A cesarean section was done in an attempt (successful) to at least save the infant's life. The others died four days, five days, ten days, and eleven days postpartum, with signs of increasing exhaustion of the heart muscle.

In this group the striking feature of the case reports is the evidence that it is the pregnancy rather than the labor that does the damage; 14 of the cases were multiparæ with rapid easy labors, yet all showed a tendency to collapse afterward. More-

over, those whom we had an opportunity to watch antepartum showed evidence of a deficient cardiac response, as indicated by a constantly rapid pulse, breathlessness, etc. As already mentioned, 14 had rapid easy labors; 7 were assisted by forceps (including 2 in whom an induction was done); 3 had version and extraction performed, and 4 had cesarean sections.

One of the cesarean sections was the case moribund on admission already dealt with; the other 3 were operated on for pelvic obstruction in spite of, not because of, the heart condition; 1 was for a flat pelvis, 1 for a large fibroid, and 1 for an associated carcinoma of the rectum. The last died four days after operation, the others did well.

The histories of 2 cases illustrate the danger of allowing pregnancy to take its course in this group while awaiting evidence of *serious* breakdown. In this it is in contrast to Group 1 with mitral stenosis.

The first case came to hospital when six month's pregnant in a very grave state of heart failure, as evidenced by marked cyanosis, dyspnea, and edema of the limbs. She was thirty-two years of age and pregnant for the second time. She was immediately taken in hand, given all the help possible, but five days later died undelivered, having failed to respond in the least to any treatment.

The second, a young woman of twenty-two, second pregnancy, came to see me when ten weeks' pregnant, having returned to Montreal because of breathlessness, palpitation, and precordial pain. Her pulse was 140 and she looked very ill. She was immediately seen by a member of the staff of the hospital and her uterus emptied the same week, with a very immediate improvement in her pulse and symptoms. The procedure in this case was justified by subsequent events. She developed acute inflammatory rheumatism four months later while on a visit to Quebec, and died of heart failure within one week of its onset. It seems hardly possible that had her pregnancy been allowed to proceed she would have been able to survive it.

7. A small group of cases with *unusual cardiac defects* is of more interest than importance. It includes 2 cases of congenital

heart lesions and 5 cases which have been classified in the records as aortic stenosis.

The first of the congenital cases was immediately admitted for observation when she applied at the Outdoor Department. She had an appearance typical of a case of pulmonary stenosis: marked cyanosis, clubbing of the fingers, etc., and a rough rasping murmur over the pulmonary cartilage. She gave a history of two deadborn babies. While under observation, as well as at the time of labor, she showed no signs of cardiac distress. An induction with version and extraction was done, chiefly because of the loss of her previous children and to make certain of a living child. The second one, a primipara twenty-five years of age, with a similar lesion, was first seen three weeks before confinement. At that time the following note was made on her preliminary history sheet: "There is a systolic murmur present at the base due to a congenital condition, and it is not thought that it will be affected by or affect labor." Subsequent events proved the prognosis to be correct, for she had a normal easy labor without any apparent effect on the heart.

That we should have 5 classed as *aortic stenosis* in a series of 159 cases is out of all proportion, but the diagnosis in every case was made by one of our senior physicians. The explanation is to be found, I think, in the rarity and the obtrusiveness of the lesion; when the patient presented herself to the practitioner, he referred her to hospital because of his uncertainty as to the outcome. That aortic stenosis is not to be dreaded from the obstetric point of view seems clear from our cases. The first was twenty-five years of age and pregnant for the second time. She showed no distress before or during labor. The second was forty years of age and pregnant for the seventh time. Although there was some dyspnea, which caused her to be sent in antepartum, she did well and left the hospital in good condition on the fourteenth day. The third, like the second, was forty years of age and pregnant for the seventh time, and was sent in antepartum for early signs of cardiac failure. She also did well. The fourth was pregnant for the ninth time. All

her previous labors had been normal. On admission she was in a condition of broken compensation with evidence that the myocardium was involved. After five weeks' rest in bed an induction was done and a small child removed by forceps. There was rapid improvement after her labor, but she broke down again a few days after she left the hospital, and she died in the General Hospital one month postpartum as a result of the associated myocarditis. The fifth case, thirty-two years of age, pregnant for the second time, also had an associated myocarditis. She had previously been under observation in the Royal Victoria Hospital. She had a normal easy labor, but collapsed at the end of the third stage; she responded rapidly to stimulation and left the hospital in fair condition two weeks later. These 5 women with aortic stenosis had 27 children, and it was only when the myocardium became affected by multiple pregnancies that any disturbance resulted.

The following conclusions appear justifiable from a study of these cases:

1. Certain heart conditions are very adversely affected by pregnancy, and it constitutes for them a very real complication. Included in this group are auricular fibrillation, myocarditis, and mitral stenosis.

2. Marriage and pregnancy are not justifiable in cases of auricular fibrillation or myocarditis as here defined.

3. Should any woman after marriage develop either of these conditions and subsequently become pregnant, she should be aborted and sterilized without awaiting signs of breakdown.

4. Cases of mitral stenosis with no evidence of myocarditis or of previous heart failure are capable of bearing one or two children with safety if carefully watched, but each pregnancy damages the mother's heart very considerably.

5. With close attention to the signs and symptoms of heart failure as evidenced by breathlessness, palpitation, precordial pain, and râles at the bases of the lungs one can forestall a breakdown in cases of mitral stenosis by interference, either by induction or by section, according as the special case may indicate.

6. Unless specially indicated, abdominal cesarean section is

not as satisfactory a method in cases already showing some heart failure as induction of labor or vaginal section, leaving the sterilization of the patient to some subsequent occasion.

7. Certain other heart conditions are not much affected by pregnancy, and there is no need to advise against marriage on account of the lesion itself. This group includes mitral regurgitation, aortic regurgitation, aortic stenosis, and the congenital lesions.

8. Finally, it is evident that it is the *heart muscle* that is the factor of importance, and that all other factors are of consequence only in so far as they may involve it.

It was on the strength of these conclusions therefore, gentlemen, that we advised this patient as we did three months ago and we still feel that this advice was warranted.

CLINIC OF DR. COLIN K. RUSSEL

ROYAL VICTORIA HOSPITAL

ON THE NATURE OF TABES

FEW disease conditions offer such interesting problems for study as does tabes dorsalis. It is so relatively common, the signs are so obvious and well recognized, and yet one cannot find in any text-book any completely satisfying explanation of the complex of this disease.

The explanation of the ataxia is, of course, obviously the loss of sense of position in the muscles secondary to degeneration in the posterior columns; the lightning pains, the girdle sensation, and the various visceral crises are satisfactorily explained by irritation of the sensory roots by foci of syphilitic meningitis, and possibly the trophic disturbances, such as the perforating ulcer and the bone changes that are sometimes present, have also likewise an explanation in degenerative changes which have taken place in the peripheral nerves as described by Mott. But there are a great many other facts in the symptom-complex of tabes which are not so readily explained; for instance, the Argyll Robertson pupil—the most common sign in this whole symptom-complex, a tabetic symptom—if one may use the expression—which is not confined to tabes, but may be present in any long-standing infection of syphilis where the nervous system has become involved. Again, the optic atrophy may possibly in a few cases be caused by a gummatous meningitis of the base, but in the great majority of cases this is not so, and an interesting and well-known fact is that where optic atrophy, with blindness, occurs, ataxia does not develop, and in many cases where blindness due to optic atrophy supervenes in a patient who is already ataxic there is a very definite tendency toward improve-

ment in the ataxia, and this in spite of the fact that the ataxia was worse when the eyes were closed.

The hesitancy in starting micturition in tabes is a well-recognized symptom, and it is also well known that this not infrequently goes on to development of incontinence, but in my experience, previous to this stage of development one practically uniformly gets the history of the individual being able to go for twelve to twenty-four hours without any desire to empty the bladder.

Some of the other interesting problems in tabes arise from the less commonly known facts. The disease is certainly more common in men than in women. Mendel, among his private patients, found the relative incidence as 25 men to 1 woman, whereas in his public clinic the incidence was 3 men to 1 woman. It used to be a commonly recognized fact that there was always an outcrop of locomotor ataxia among soldiers in time of war, and I myself remember when in England, in the hospital, being impressed with the number of cases that dated the onset ot their symptoms to service during the South African War. I venture to say that in this recent war, on the other hand, there were 20 cases of general paresis for every 1 of tabes. These and other facts which will develop as we proceed are the facts which any satisfactory theory of the pathogenesis of tabes must explain.

The General Pathology of Tabes.—One can say now, without fear of contradiction, that tabes and taboparesis are practically always due to syphilis; possibly in the odd case one may have doubts. One finds evidence of this infection not only in the nervous system, but outside as well, in the vessels particularly; gummata of the bones or in other organs are not uncommon, but the striking thing about tabes is that its symptoms usually develop many years after the primary infection, and the degeneration in the cord has not the characteristics of a syphilitic change. The spinal cord shows a degenerative lesion of the posterior columns and posterior nerve roots which has gone on to sclerosis. The morbid process selects first *certain groups of fibers* in the posterior columns, and spares others, but eventually in advanced cases may destroy all the exogenous fibers. In

some cases where the lower extremities have been affected the lumbar and lower dorsal part of the cord bear the brunt of the disease; in others, the so-called cervical tabes, these parts may show relatively little degeneration, whereas the cervical part of the cord shows a very marked sclerosis.

The Posterior Roots.—Microscopic examination almost invariably shows degeneration which is relatively proportionate to the atrophy in the posterior columns. There is, according to Mott, degeneration of the coarse fibers entering the cornu radicular zone and proceeding respectively to (1) the root zone of Charcot, (2) to the cells of Clarke's column, and (3) to form the column of Gall. There is almost invariably a degeneration of the fibers of the posterior nerve root approximately proportionate to the atrophy of the exogenous fibers in the posterior columns. The changes in the cells of the spinal ganglia, on the other hand, are very insignificant as compared with the degenerative atrophy of the fibers emerging from the ganglia. Some cells show an atrophic decay, but the majority appear fairly normal with the methods at our disposal. Mott observes that in longitudinal sections of the posterior spinal ganglia, with a long attachment of the ventral and dorsal roots, the myelin sheath of fibers which still possess the property of staining blue by the Pal or Weigert staining, becomes sometimes more attenuated and fainter as one proceeds away from the ganglion toward the cord, the peripheral fibers proceeding from the ganglion remaining quite healthy in appearance as regards myelin staining, in an advanced case of tabes, and yet the remote peripheral sensory fibers show degeneration. Nageotte and, more recently, Richter[1] look upon the degneration in the exogenous fibers in the posterior columns as due to involvement of the posterior nerve-fibers in a syphilitic granulation tissue in the lymphatic spaces of the combined dural arachnoid sheath. From there the granulation tissue, according to them, makes its way into the looser tissue of the subarachnoid space and invades the nerve bundles. Obersteiner and Redlich think the constriction takes place where the nerve root penetrates the pia and where the myelin sheath

[1] Zeitschr. f. die gesamte Neurol. und Psychiatrie, vol. xlvii, p. 1, 1921.

of the nerves is not so well developed. They add also further constriction by arteriosclerotic changes in the posterior spinal artery. Mott criticizes this theory on the grounds that in 11 of the 28 cases which he examined, he found the fine fibers of Lessauer's tract but slightly affected, whereas the cornu radicular zone was much affected. He points out that if meningitis were the cause of the degeneration it should not be selective in sparing the fine fibers of Lessauer's tract. It is obvious that such a mechanical theory cannot satisfactorily explain why in some cases the lumbar region is involved, and in others the cervical region, although the meningitis may be present in both regions. It is difficult to explain also why the anterior nerve roots should be spared. No such mechanical theories can begin to explain the tabetic picture.

The Stress Theory.—Weigert and Roux have shown that the various constituents of a tissue are normally in a state of equilibrium so correlated one to the other that no cell can disappear without the surrounding tissue growing to take its place, and when one constituent becomes weaker or less resistant, the energy of growth of its neighbors repress it still further.

Function requires consumption of part of the active tissue, this normally is compensated for by sufficient supply of suitable nutrition, otherwise the normal equilibrium of the parts is disturbed and a progressive degeneration will result. Edinger assumes that if the supply of nutrition be deficient, or if, though it be normal, excessive work will be demanded of the cell, that is, if the normal relation between combustion and repair is disturbed by either relative or absolute superfunction, the energy of growth of resting tissue will bring about a degeneration of the less resistant active parts, and this result would more easily occur if both possibilities are combined and excessive work be associated with a state of nutritional deficiency.

Voss, by injecting pyrodin in animals, hoped by producing an anemia to bring about changes in the cord such as one finds in severe anemias. Although he failed in ill-nourished animals which he nursed to keep alive, Edinger and Helbing succeeded in producing degeneration in the cord very similar to what one

sees in tabes by subjecting these experimental animals to the stress of overwork—function evidently was the determining factor in producing the degeneration—Voss's experiments really being a control to Edinger and Helbing's work. There are undoubtedly a small number of nerves which are constantly or very frequently at work or, at least, much more constantly in action than any of the other nerves of the body. These are the sensory nerves from the muscles which take an important part in the regulation of muscular contraction, and are constantly transmitting those stimuli by which we can become aware of the condition of our muscular system or the position of our limbs. These nerves are almost constantly at work, and if the hypothesis is correct, these must suffer and their degeneration will be clinically expressed by loss of muscular tone and loss of sense of position—ataxia—and, of course, if an individual's occupation made it necessary for him to use one limb or one pair of limbs much more than the other pair, one would expect the ataxia to express itself first in the limbs most used.

There is not merely the question of toxemia, but in the presence of a slowly acting fairly virulent toxin *function creates the symptom-complex*. As Gordon Holmes has pointed out, the spinal changes found in some cases of severe anemia or occasionally associated with diabetes or in the marasmic state brought about by the poisons ergotin and diseased maize have undoubtedly a close resemblance to the tabetic lesions, differing chiefly in their more rapid course and less regular localization. Holmes has also pointed out that one would not expect the anterior horn cells to be affected if one considers the peripheral neurons, afferent and efferent, and the corresponding pair of upper or central neurons, that is, the pyramidal tracts and the secondary sensory fibers. Unquestionably far greater stress falls on the latter. The pyramidal tracts are only active at times, and may have long periods of rest when they may compensate any of their material which was used up. "The peripheral motor neurons may be kept in action by stimuli from two different systems—an interrupted activity, the result of stimuli from the

pyramidal tracts, and a constant activity, represented by muscular tone dependent on impulses carried to them by the corresponding peripheral afferent nerves. Thus the peripheral motor nerve which is normally capable of responding to two sets of stimuli, when exhausted, has abundant opportunity to recoup during the periods when it is not at the service of the voluntary impulses, but with the peripheral sensory neurons and, more especially, the afferent muscular fibers it is different. Their activity is constant and unremittent—they keep our motor mechanism informed of the position of our limbs and complete the reflex arc which extends from skin to muscle."

In syphilitic infection the more virulent and active types run to well-marked skin eruptions and granulomatous lesions of the various viscera, including the brain and cord and their membranes. The less virulent and active are more likely not to produce secondary eruptions and manifestations to the same extent at least, but in their insidious growth prolonged over a much greater length of time—as they do not produce symptoms that bring the patient for advice—their toxic affect has sufficient time to manifest itself. It has long been recognized that in a very large proportion of tabetics no history of secondaries can be obtained.

It is certain that on this basis Edinger's dictum, "In tabes function creates the symptom-complex," gives a far more satisfactory explanation than has yet been offered for this complicated picture. Some years ago I was able to demonstrate the truth of this experimentally by taking rabbits which had been inoculated with syphilis, and by exposing them to rapidly alternating light and darkness produced an immobility of the pupils to light.

Case I.—S. C., male, aged thirty-five, by occupation chef on a dining car, complains of failing vision, sudden sharp shooting pains in the legs and back, and some difficulty in walking in the dark.

His history is as follows: He contracted lues twelve years ago and was treated for five months by hypodermic injections

of mercury. Fourteen months ago—*i. e.*, before admission to the clinic—he first noticed failure of vision, which progressed till, on admission, he could read only the heading of the newspapers with the left eye, and he was blind in the right eye. For four months before admission he noticed some impaired sensibility of the bladder, being able to go for twenty-four hours without desire to empty it; he has now some difficulty in starting the stream. There has been some difficulty in walking in the dark, and there have been definite lightning pains.

Examination.—There is bilateral optic atrophy, causing blindness in the right eye and very defective vision in the left, he can count fingers at about 2 yards; the pupils are inactive to light, but react to accommodation. There is a scarcely perceptible ataxia of the lower extremities evidenced in touching the heel to the opposite knee, and a slight impairment of the sense of position of the muscles, which is shown by his not knowing in which direction one moves his toes if his eyes are closed when one is doing it. The knee- and ankle-jerks are absent. Romberg's sign is present. The patient's condition has not altered perceptibly since his first admission, save that for a time, at the end of last winter, it was noticed that there was an increase in the loss of the sense of position in the lower extremities. This was accounted for by the fact that he used to walk into the clinic from one of the suburbs every week or two, and the walking was very heavy.

It is evidently a typical case of tabes with optic atrophy.

Now, on going into his history more carefully, it is found that while employed on the dining car he was in the habit of reading for two hours every afternoon and again from 9 to 12 at night, usually lying down in his bunk, by the light of a coal-oil lamp, while the train was running along. The strain on his eyesight can be imagined. It is interesting to note that impairment of vision was for a long time his only complaint, and especially that since he has given up all occupation that would cause any strain on his sight this has not become any worse.

It is common knowledge, as has already been mentioned, that when optic atrophy causes blindness ataxia does not develop.

This is probably explained by the fact that a blind man cannot get about enough to throw the necessary stress on the nervous system by which ataxia is produced.

Case II.—G. P., male, aged thirty-three, by occupation a tailor, complains of disturbance of vision, weakness in the arms, and "electric shocks" in the arms and legs, occasional vomiting. He gives a history of a primary luetic infection eleven years ago. His present trouble began three years ago, with disturbance of vision, and the eyes became crooked as they are at present; about the same time he began to suffer from pins-and-needles sensation in the arms, and he had increasing difficulty in carrying on his occupation of sewing. Some time later he developed these sensations in the legs also. He states that he empties his bladder about once a day as a rule.

Examination.—The fundi are normal, there is marked bilateral ptosis with compensatory contraction of the occipitofrontalis in the endeavor to hold up the eyelids, there is an outward deviation of the eyes due to a weakness of both internal recti; he can fix either eye on an object to the nasal side, but there is marked secondary outward deviation of the other eye on the attempt. There is a slight impairment of upward movement of the right eye.

The right pupil is larger than the left, and neither react either to light or to accommodation. There is very marked loss of tone in the upper extremities, but no actual loss of power; the use of the arms, however, is interfered with by an extreme ataxia, especially on the right side (it might here be noted that he is a right-handed man); the loss of tone in the lower extremities is scarcely perceptible, and, as you see, there is little or no ataxia in walking. There is an extreme loss of the sense of position in the upper extremities; if with his eyes closed he is told to touch one thumb with the finger of the other hand, he can only do so by finding first the elbow and following up the limb from that. There is delayed sensibility to pain in the lower extremities, and to a lesser degree in the upper. The knee- and ankle-jerks are absent.

On inquiring into this man's occupation, we find it was to sit and sew all day, threading his own needle. We have, then, immediately the explanation of the location of the disease in this case. "Function creates the symptom-complex." Here we have, then, not the usual and simple loss of the reaction of the pupils to light, but owing to the patient's occupation necessitating the frequent use of the reflex of accommodation in order to thread his needle, the neurons governing this movement have also become affected, and we have the loss of reaction of the pupil to accommodation, and paresis of the internal recti. In the same way the reason for the affection of the arms is obvious, and the escape of the lower extremities.

Case III.—A. C., male, aged forty-four years, by occupation a cigar roller, complains of blurred vision for the last two years, and some difficulty in walking in the dark for six months past; he has had shooting pains in the legs for the last thirteen years and girdle sensation. He frequently passes a day without the desire for micturition and has difficulty in starting the stream.

Examination.—There is bilateral optic atrophy, the pupils have lost the reaction to light, but react to accommodation. There is a slight but definite ataxia in the upper extremities, especially marked in the right arm, and a very definite loss of tone in the muscles; the ataxia is also present, but to a less extent in the lower extremities; the knee- and ankle-jerks are absent. Romberg's sign is present.

On looking into his history more carefully, we find that previous to two years ago his place at work was in a very dark part of the room, and he says it was a great strain on his eyes to see well enough to finish off the ends of the cigars; since that time he has his seat in a better lighted part of the room near the window.

Case IV.—A. S., male, aged thirty-six, a shoemaker, complains of inability to walk and lightning pains.

For three or four years the patient has been in the habit of congratulating himself on his ability to hold his water, even

making it a matter of boasting that he could go from the time he got up until he went to bed again without emptying his bladder. In April, after selling his place of business, he started out with what was left to him in a bullock wagon, and walked for eight days and nights across country, in cold and rainy weather, becoming terribly fatigued, and was "snow blind" for weeks after this; from that time he has been unable to walk without support, not being able to control the movements of the legs; he cannot attempt to walk in the dark. In May he found that he could not urinate when he wanted to, and he began to have incontinence. He has had the lightning pains for the past seven months. There is a history of a chancroid ten years ago, but there were no secondary symptoms.

Examination.—The fundi are pale and atrophic looking, there is the typical Argyll Robertson pupil, and internal strabismus due to a paresis of the left external rectus. There is marked loss of tone and extreme ataxia of the lower extremities, so much so that the patient cannot walk; he has impairment of sensibility over the feet and about the mammary regions. The knee- and ankle-jerks are absent.

Following the other cases the explanation of this one is obvious. Of course the snow blindness was simply a conjunctivitis, but the glare of the snow on the eyes was quite sufficient to cause a partial optic atrophy, and the journey, exposed as he was to wet, cold, and extreme fatigue, sufficient to produce exhaustion and determine the symptoms in the lower limbs.

Case V.—I would like to quote another case, a man aged forty-four, with typical tabes, the lightning pains and the ataxia, the Argyll Robertson pupil, and loss of tendon jerks; he said he was able to hold his urine for twelve hours without distress, and had some hesitancy in starting micturition. He had had lues twenty-five years ago. He stated quite spontaneously that for some years his duty had been to go and clear up any wreck that occurred on the railroad, and, of course, this work had to be done quickly. On many occasions he would be on his feet for thirty-six hours or more, working strenuously, and he

had frequently noticed that at the end of this time, when he was tired, he would have unsteadiness and difficulty in walking similar to that present at the time of examination, but that this would pass away in two or three days with rest. His ataxia had really begun only two months ago, after a severe grippy cold, combined with a certain degree of mental worry and distress owing to the illness and final death of his mother.

In studying the ataxia in any of these cases where the lower extremities are involved it will be obvious that it is not confined to the legs alone. It is only necessary to get the patient to sit down, stand up, go up or, especially, downstairs, and one will recognize that the trunk muscles also are involved, as one would expect. In walking, the center of gravity of the trunk is far behind where it should be, and the same thing is even more noticeable in the other movements.

The Argyll Robertson pupils, a symptom common to all these cases, is explained by the fact that the reaction to light is a reflex act, and not being under the influence of the will, is more constantly active with every glance from light to shade, whereas the reaction to accommodation is a voluntary act, and, therefore, not so commonly used. The greater stress is obviously on the neurons, which subserve the reaction to light producing exhaustion. This symptom may be the only tabetic symptom present in a case of syphilis of the nervous system.

It is noticeable also that in all these cases the bladder symptoms are identical, first, the exhaustion of the sensory neurons producing loss of sensibility; this gives rise to distention and loss of tone shown by the hesitancy in starting micturition. Then the added work thrown on the sphincter finally causes exhaustion of its nervous mechanism and incontinence develops. My own experience confirms that of Edinger, that where the patient is taught to empty his bladder at stated periods incontinence does not develop.

In the last case one sees the result of relative overactivity in producing ataxia which apparently cleared up during the intervening periods of rest, and the similar result of a superadded toxemia of an acute infection.

Now if we consider Mendel's statistics, which, by the way, are not isolated observations, he found tabes less than three times as frequent in males than in females among the patients in his Policlinic, whereas in his private practice he found 25 males to 1 female. This is probably not due to any greater incidence of syphilis among the women in the poorer walks of life because it is noticeable that one does not find tabes among prostitutes. Women of the more wealthy classes, such as would make up his private cases, are not exposed to the influences which produce tabes, neither are the frequently luxuriously living prostitutes. Whereas the nervous systems of women of the lower classes and from the slums, fully exposed to all the wear and tear of life, would be likely to succumb to the undue stress, and it is not surprising that they should suffer almost as frequently as the men from tabes.

It has been shown that tabes is best regarded as an elective radicular degeneration, that is, that the degeneration in the posterior columns is not in mass, in the early stages at least, but in groups according to function. This, while difficult to explain by any other theory, is readily explicable by the stress theory. The fact that the degeneration is practically confined to the intramedullary part of the sensory neurons, although the peripheral parts are exposed to the same stress, may well be due to the increased resistance given to the peripheral part by its neurilemma sheath, we know the intramedullary part has not the same power of regeneration as the peripheral nerve.

Treatment.—Understanding tabes from this point of view we have very definite indications for treatment, at least prophylactic. These indications are, first, limit as far as possible the production of the virus by whatever specific treatment may be indicated. It is common experience that certain patients are not benefited by specific treatment, in fact, may be harmed by it. In my experience if the cell count in the cerebrospinal fluid is within normal limits, and is not raised after an intravenous injection of novo-arsenobenzol, specific treatment is not indicated, always, of course, taking it for granted that there is no indication for treatment outside the nervous system,

therapeutic measures in that case should be directed toward increasing the general health and resistance.

For the ataxia, re-educative exercises, as laid down by Prof. Fränkel, will perform wonders if carried out with care under proper supervision. Here again the stress or exhaustion theory is, in my experience, of real help; and the results of treatment rather justify the theory. If the patient be allowed to carry out the exercises without proper supervision, in his enthusiasm to get well he may be tempted to practice too long and at too frequent intervals, with the disappointing result that he will get worse instead of better. To avoid fatigue it is necessary to watch the pulse. At first, after carrying on the exercises for only a minute or two, it will often be found that the pulse-rate has increased 20 or more beats to the minute; this indicates fatigue, and the exercises should be stopped until the pulse-rate has slowed down again, when they may be resumed. Carried out with this supervision the results are good.

CLINIC OF DR. L. M. LINDSAY

CHILDREN'S MEMORIAL HOSPITAL

NUTRITIONAL EDEMA

WE have for consideration today the subject of edema as it occurs in the course of nutritional diseases, especially of infants and children. This form of edema is occasionally seen in infants who have suffered for some time from malnutrition, and also in those who have been kept for too long a time on cereal decoctions for the treatment of diarrhea. In both cases the edema is quite independent of any disease of either the heart or the kidneys. It is, therefore, entirely due to derangement of water metabolism within the body.

The edema appears first in the feet and hands and may spread over the whole body. It is usually confined to the subcutaneous and muscular tissues, and rarely occurs as free fluid in the large serous cavities. The skin over the affected part is tense and pale and pits on pressure. The first indication of an on-coming edema is often an unaccountable gain in weight.

During the first year of life there seems to be a greater tendency for edema to develop, which diminishes as time goes on. This tendency is probably favored by the liquid diet and the frequency of nutritional disorders in the first year. Yet no age is absolutely immune, for we shall see that even adults under certain conditions will develop dropsy as a result of faulty diet. According to Czerny, certain infants are endowed with a "dropsical constitution," which means that they retain and discharge an abnormal quantity of water under slight provocation.

In considering the pathogenesis of nutritional edema we have to bear in mind certain fundamental principles of water metabolism. Wherever water occurs in the body there are

present at the same time colloids, particularly proteins and crystalloids, such as salts and sugars.

The relation of inorganic salts to the movement of water in the organism is very intimate. Sodium chlorid has the greatest power for producing edema. Bicarbonate of soda is less potent, while potassium and calcium salts have least effect. Indeed, there is evidence to show that calcium may, under certain conditions, hinder the transudation of fluid into the tissues. Still reports the case of an infant who was given 10 grains of sodium citrate in each feeding, with almost immediate appearance of edema. When the salt was discontinued the edema rapidly disappeared, only to reappear when the salt was added again. It is probable that young infants have not the same power of excreting salts as older children, but in the type of edema we are considering mineral salts play but a secondary part and merely follow the distribution of water.

Carbohydrates have a similar though less potent effect on the retention of water. This is explained on the grounds that glycogen or glucose has the power of binding two or three times its own weight of water. This water is, however, loosely combined, so that it is easily and quickly discharged.

Clinically we often see the result of this action of carbohydrates on water retention as evinced by the weight curve. Infants fed on proprietary infant foods, which consist largely of starch and sugar, may be far above the average in weight, but they are pale and flabby and have a low resistance to infection. It is these infections which often determine a sudden discharge of water from the tissues, with a corresponding drop in weight.

Closely allied to the foregoing is a peculiar type of nutritional disturbance occurring in infants as a result of feeding undue quantities of flour. This condition is not so common in this country as in Europe, where it is known as Mehlnahrschaden. One of the characteristics of this disorder is the great and sudden variations of the weight curve, which may occur spontaneously or as the result of bacterial infection. Edema is not infrequently seen in the course of this disorder.

Occasionally one sees an infant whose diet would seem to be sufficient and complete, and yet whose weight remains stationary. If now one adds to the diet a small amount of barley flour or cream of wheat, the weight immediately begins to rise, and from this time the child continues to gain. This demonstrates the beneficial effect of water retention induced by carbohydrates.

In 1914 Potter observed a number of cases of infantile diarrhea in which edema developed on a diet of barley-water, and found that if protein and fat were added to the food the edema disappeared in a short time. He also reported another group of atrophic infants who developed edema while on a diet of whey and barley-water. This also cleared up when protein and fat were increased in the food. He, therefore, came to the important conclusion that the edema in both these groups was due to a deficiency in the diet of some essential element, probably protein or fat.

That other deficiencies in the diet may lead to dropsy is now well known. Edema is said to constitute the main symptom of ship beriberi. Infants nursed by beriberic mothers develop edema as their most constant symptom, and it is not uncommonly seen in Barlow's disease. In each of these the edema will promptly subside when the specific vitamin is added to the diet.

A new impetus was given to the study of nutritional edema during the recent war, when so-called "famine" or "hunger" edema made its appearance in war-bound countries. Many articles have been written on the subject, and as many theories advanced to explain its pathogenesis. The thin soups deficient in protein and fat, the lack of fresh fruits and vegetables, underfeeding, overwork, and the exposure to cold, were each in turn or in combination held responsible for the dropsy. An important discovery was made in the blood, which was found to be not only hydremic but also poor in proteins and lipoids.

In health the serum proteins are maintained at a fairly constant level, and any change is taken to indicate a difference in the water content of the blood. It is well known that serum

proteins exert a great influence on the osmotic pressure of the blood, and that any diminution in their concentration results in a lessened ability to hold the water within the vessels, and so favors the imbibition and retention of fluid by the tissues.

Here, then, is at least a partial explanation for the development of edema, which may be facilitated by an increased permeability of the capillary walls resulting from the undernourishment. With the retention of water in the organism there is naturally retention of sodium chlorid, though the latter is found to be present in normal amounts in the blood.

These and other investigations lead to the conclusion that deficiency of protein in the diet is the main if not the only cause of the edema. This conclusion has been substantiated by animal experiments carried out by Kohman, who found that a large percentage of young rats developed edema if fed on diets in which carrots were the only source of protein—all other essentials being supplied in the food. If an adequate protein-like casein were added to the diet the edema could be prevented or cured. Doubling the salt ration had no apparent effect on the production of edema, which, however, did occur more frequently on a wet diet.

It is obvious, therefore, that war edema in adults is a very similar if not identical disorder to the nutritional edema of infancy. It is apparently caused by the same defect in the diet and can be cured by the same means.

Utheim has shown that the serum protein of normal infants varies from 6 to 6.5 per cent. during the first year, and this rapidly rises during the second year to reach the adult level of about 8 per cent. Premature and athreptic infants show a constant diminution in the serum protein, which may fall as low as 4 per cent. This may partly account for the appearance of edema which is sometimes seen in premature and debilitated infants.

While the prognosis is grave when edema appears in infancy, it is not necessarily hopeless, nor is it so serious an omen as purpura. In older children and in adults the outlook is much

less serious provided the undernourishment has not lasted sufficiently long to damage the organism irrevocably.

We may now apply the foregoing considerations to the prevention and treatment of edema in infancy and childhood. As the edema is a symptom or sequel of some profound disturbance of nutrition, it naturally follows that our attention must be directed to the underlying condition.

In treating a case of marasmus or diarrhea one must not underfeed the infant for more than a few days. At the onset of an acute digestive disturbance it is often advisable to starve the infant for twelve to twenty-four hours—only giving a thin cereal decoction. Then protein milk or lactic acid milk should be added in suitable quantities irrespective of the nature of the stools. Both of these foods are relatively rich in protein and low in sugar, and protein milk is also low in salts. Whey, on the other hand, is the reverse of all this, and should be avoided.

In this connection it would be interesting to know if the occurrence of edema were less frequent during the past decade since the introduction and more general use of protein milk in the treatment of digestive disturbances of infancy.

In the more chronic nutritional disturbance known as marasmus or athrepsia the diet should contain a sufficient amount of the essential elements of food, even if some of these are reduced to a minimum. If whole milk mixtures are not well tolerated and breast milk is not obtainable, one can usually succeed with lactic acid milk or powdered milk suitably modified.

When edema has actually developed we know there is an advanced degree of athrepsia, and only by the most careful treatment can the infant's life be saved. Although breast milk is considered the ideal food for the disorder, I have sometimes found that breast milk combined with protein milk (3 to 1) is better tolerated and more efficacious.

While the treatment of athreptic infants with edema is almost entirely a problem of feeding, certain other therapeutic measures are of undoubted assistance in tiding the infant over so critical a period. Perhaps the most important of these is

blood transfusion, which will, at least temporarily, benefit the infant, and may be the turning-point in the course of the disorder.

I have also found caffein to be of value, as might be expected, for, besides being a stimulant to the circulation, it increases the output of both salt and water through the kidneys. Finally, it is claimed that the action of caffein is enhanced by

Fig. 320.—The patient as he appeared six months prior to admission; fat and flabby, but no definite edema.

adrenalin, which not only causes diuresis but also facilitates in some way the absorption of fluid from the tissues.

The following case illustrates some of the problems we have been considering:

A Jewish boy of four and a half years was admitted to the Children's Memorial Hospital complaining of swelling of the legs and puffiness of the face. All his life he had been "a difficult feeding case" and for the past three years had attended

our Out-patient Department more or less regularly for the treatment of chronic intestinal indigestion with infantilism, as described by Herter. His progress had always been irregular, partly owing to lack of co-operation on the part of his mother and her inability to follow the diets prescribed. A photograph taken six months before his present admission shows him to be enormously fat, and the note made at that time states that he was pale and flabby, though there was no definite pitting

Fig. 321.—The same patient on admission, showing marked edema and dilated venules.

on pressure. His diet is said to have been chiefly biscuits and bananas, as he refused everything else.

About one month prior to admission swelling of the feet and legs was first noticed. This gradually increased in amount and spread to face, hands, and back. His diet at this time consisted largely of biscuits, cocoa, and oranges, with occasionally soup and eggs. On admission at the age of four and a half years he weighed 25 pounds and was $31\frac{1}{2}$ inches tall, so that even with the edema he had the height and weight of only an eighteen-month child. Figure 321 shows his appearance on admission

and the extent of the swelling of the extremities. These were not only greatly swollen, but cold and marbled, with a brownish network of distended vessels. The swelling of the face, hands, and genitalia is not so apparent, though it was well marked. There was impetigo about the mouth. No fluid could be detected either in the thoracic or abdominal cavity. The abdomen was large and distended, as is usual in these cases. His temperature per rectum was 99.5° F. Nothing abnormal was made out in the heart. Analysis of the urine was as follows:

Clear orange, 1030; acid; a trace of albumin, no sugar, no acetone, no indican, no pus, no casts, and no erythrocytes.

Examination of the *blood* showed the following:

Red cells, 3,000,000; leukocytes, 9700; hemoglobin, 35 per cent.; serum protein, 5.1 per cent. (normal 8 per cent.), and blood chlorids, 0.6 per cent. (*i. e.*, normal).

Thus, besides the marked anemia, the blood is normal in chlorids and very low in serum protein.

The edema was, therefore, considered to be entirely due to his nutritional disturbance.

Treatment.—He was kept warm in bed and an effort made to get him to take sufficient food. The diet consisted chiefly of lactic acid milk, junket, gelatin, scraped beef, with strained vegetables and farina. Orange juice and cod-liver oil were also given. Caffein was given in the form of tea twice daily, as he was evidently accustomed to this beverage. Fluids were moderately restricted and no added salt was allowed.

During the first fortnight in the hospital his weight dropped from 25 to 19 pounds, and the edema disappeared. Although his appearance was worse, his appetite had improved, and his stools were good. The serum protein had now risen to 6 per cent. and it remained at this level for some weeks.

Convalescence was naturally slow, but he gradually improved and steadily increased in weight. Now, after six months, he is still pale, weighs only 31 pounds, but has had no return of dropsy.

CLINIC OF DR. F. G. FINLEY

MONTREAL GENERAL HOSPITAL

MITRAL STENOSIS WITH VENOUS THROMBOSIS

GENTLEMEN: I am bringing before you today 2 cases which you have seen in the wards to illustrate the clinical course of mitral stenosis, both of them being complicated with venous thrombosis.

Mme. F., aged forty-one, presser, was admitted to hospital on November 30, 1923 complaining of shortness of breath, cough, pain in the liver region, and much loss of strength.

She was in hospital for inflammatory rheumatism at the age of six and states that she has had a rheumatic heart since that time. As a child she was in poor health, unable to attend school, and spent much of her time in bed under medical care. She has, however, had no further attacks of rheumatism, nor has she suffered from recurring attacks of tonsillitis or other infections common in childhood. At fourteen her health began to improve. She married at twenty-five and had 3 children and 3 miscarriages, her confinements being unattended by any difficulty. Her last child was born three years ago, and since then she has suffered from shortness of breath on exertion and profuse menorrhagia and metrorrhagia, bleeding being present on about twenty days each month.

In the family history the only point of interest is that her mother suffered from rheumatism and died at fifty-three, presumably from heart disease, after being an invalid for many years.

Seven weeks before admission she had a severe sore throat and high temperature—probably acute tonsillitis. After a

week in bed she tried to resume her work, but was too feeble to do more than half a day at a time. Weakness became so pronounced that she was obliged to stay in bed for a fortnight before admission. There has been some vomiting for the past week.

On examination she is a fairly well-nourished woman, propped up in bed to breathe, and with a slight grade of cyanosis of the lips and cheeks. The mucous membranes are pale and the temperature 98° to 100° F. The thorax is well formed and a few sibilant râles are heard over the chest, with some fine crepitations at the bases.

The apex-beat is felt in the midaxillary line, with a distinct thrill. The cardiac dulness begins above at the third rib and extends $5\frac{1}{2}$ cm. to the right and $9\frac{1}{2}$ cm. to left of the midline. A rumbling presystolic murmur is heard at the apex. The pulse is small and regular. Blood-pressure 124/70.

The liver is not palpable, but there is slight tenderness below the costal border. The urine has a specific gravity of 1030, a small amount of albumin, a few leukocytes, blood-cells, and epithelium.

Two days after admission a soft diastolic murmur developed in the third left space next the sternum and along the left sternal border, and a little later a distinct venous pulse in the neck not obliterated by pressure on the upper part of the internal jugular vein. A well-marked diastolic gallop rhythm was also heard, and three weeks after admission the pulse became markedly irregular in volume and rhythm. The electrocardiogram, hitherto regular, confirmed the diagnosis of auricular fibrillation. The thrill and presystolic murmur disappeared with the onset of irregularity.

Dyspnea was not a constant feature. The respirations varied from 20 to 24, and although more comfortable when propped up, she could lie down. Accesses of dyspnea were occasionally present requiring $\frac{1}{6}$ grain of morphin for their relief. Vomiting was a prominent feature in the latter part of her illness, coming on apart from the drugs employed—morphin and digitalis. The vomitus contained a little blood, but no HCl. There was

also frequent nocturnal cough, occasional night-sweats, and for a couple of days bloody sputum, but without any signs in the lungs to indicate the locality from which it came.

Nine days before her death several hemorrhagic papules appeared on the nose, preceded by an abscess of the gum resulting from a diseased tooth. The left leg became swollen four days before the fatal termination, and in twenty-four hours the swelling and edema extended to Poupart's ligament. The pulse became weak and almost imperceptible, delirium, showing in attempts to get out of bed, developed, and she died on January 12th.

In this patient's case a diagnosis of mitral stenosis was readily made at the first examination, the apical thrill and rumbling presystolic murmur being very characteristic. The booming or accentuated first sound so often heard was only noted once during her stay in hospital, and there was no pulmonary accentuation. The diastolic murmur at the pulmonary region was at first regarded as a pulmonary incompetence, sometimes known as a Graham Steell murmur, and resulting from stretching of the pulmonary orifice. As the pulse became stronger under the influence of digitalis it acquired a slight collapsing character, and corresponding to this the pulse pressure was somewhat high, $124-70=54$. Owing to these pulse characters the diastolic murmur was attributed to a slight aortic incompetence, and, as we shall presently see, this view was borne out by the autopsy. There has been considerable discussion as to the significance of a diastolic murmur in the pulmonary region in mitral stenosis, and the question as to whether it is due to an associated aortic incompetence or to a dilatation of the pulmonary orifice from increased pressure in the pulmonary circuit is one which is often difficult to answer. Cabot found a diastolic murmur in 22 out of 50 cases with healthy aortic valves, and no dilatation of the pulmonary orifice, but offers no explanation of the sign. There is, however, reason to believe that many of these murmurs are due to a slight grade of aortic incompetence from slight damage to the valve. The pulse is the most important diagnostic criterion, and if of a collapsing

character and associated with a high pulse pressure, the murmur may be referred to such an origin.

In 50 consecutive autopsies on cases of mitral stenosis at the Montreal General Hospital acute or chronic endocarditis was present on the aortic segments in 22, and although these changes were insufficient to interfere with the functions of the valves in some of the cases, they undoubtedly led to slight grades of incompetence in a considerable number.

My own impression is that most of these soft diastolic murmurs along the left sternal border are due to slight changes in the aortic segments, and the frequency with which valvular damage is found lends support to this view.

The presence of a positive venous pulse in the neck in our case indicated a tricuspid regurgitation, regarded during life as a relative incompetence from dilatation of the right ventricle. As you will see by the autopsy report, there was an unrecognized tricuspid stenosis, a lesion which is seldom diagnosed with any certainty during life.

The considerable enlargement of the heart is not uncommon in mitral stenosis. The right ventricle is usually dilated and often hypertrophied, but it is perhaps not so generally recognized that the left ventricle is also enlarged in many instances and that atrophy is the exception rather than the rule. The early stage of mitral stenosis is incompetence, and during this stage the left ventricle dilates and hypertrophies, a condition which persists as stenosis develops.

The clinical history of this case presents several points of interest. The cardiac lesions doubtless began at the age of six, with an attack of acute rheumatism. The poor health in childhood may be regarded as a period in which compensatory changes in the cardiac muscle gradually developed, and from the age of fourteen to thirty-six she considered herself a healthy woman.

Although cases of mitral stenosis are sometimes so perfectly compensated that there is no dyspnea even on considerable effort, it is much more frequently found that this symptom is present, and its victims are obliged to restrict their activities to light exercise and avoid any form of violent exertion.

Failure of compensation in our patient, marked by dyspnea on exertion, began at the birth of her third child. Women with mitral stenosis bear pregnancy badly. The first is usually passed safely, but the second is frequently attended or followed by cardiac breakdown. Peter's dictum with regard to tuberculosis might well be applied to women with this disease, "fille pas de marriage, femme pas de d'enfants, mère pas d'allaitment." The final breakdown followed an acute tonsillitis with high fever. Next to acute rheumatism infections of the tonsil are the most frequent in originating endocarditis, and they play an important part in inducing failure of compensation in heart disease. Even with a healthy heart the cardiac symptoms known as the effort-syndrome are not uncommon after tonsillitis, as shown by the frequency of such cases in soldiers during the late war.

Symptoms of venous stasis were not prominent in this case. There was no edema of the extremities and an absence of the usual hepatic enlargement. The profuse menstrual flow, resulting from venous stasis of the endometrium, was doubtless an important factor in lessening the congestion of the right heart, and so diminishing the back pressure in the veins of the lower extremities and the liver.

Vomiting, sometimes so troublesome in advanced cardiac trouble, was rather frequent, and may be regarded as due to stasis and catarrh of the stomach, the absence of HCl from the test-meal being in keeping with this view. The lungs bore the brunt of the venous engorgment, cough and crepitation at the bases indicating edema of these organs, while the expectoration of blood in the late stages of the malady suggested hemorrhagic infarction.

Terminal infections are a frequent cause of death in cardiac patients. The hemorrhagic papules on the nose, followed by swelling of the left leg as high as Poupart's ligament, from thrombosis of one of the large veins, the common or external iliac, were regarded in this light, the focus being an abscess in the gum from a diseased tooth.

Thrombosis is an unusual complication of cardiac disease,

and when it occurs it is nearly always in the veins leading from the left side of the neck, chest, and left arm. In Dr. Welch's 24 cases it was present in the lower extremities in only 3 instances, certain mechanical factors, which will be considered later determining the localization in the upper part of the body.

The patient's age (forty-one) corresponds with the average age of death in women affected with mitral stenosis according to the statistics of Sir William Broadbent, and it is, therefore, a malady which distinctly tends to shorten life. In males the average age of death is two years younger.

Abstract of Autopsy Findings.—A small quantity of fluid is present in the peritoneal and pericardial cavities. The heart weighs 526 grams. The left ventricle shows only slight enlargement, the other cavities being dilated and their walls hypertrophied. The myocardium is soft and flabby. The tricuspid orifice is narrowed and the mitral is represented by a buttonhole orifice 2.5 cm. in length, the cusps being fused and calcified. The aortic cusps are thickened and sclerosed along their free margins. On being laid open, the measurements of the orifices are T. 5 cm., P. V. 7.5 cm., A. V. 7.5 cm., M. V. 2 cm. In the descending aorta are numerous calcified plaques. The lungs present infarcts, congestion, and edema.

The liver weighs 1170 grams and is slightly atrophied. Its cut surface presents a mottled appearance, the greater part being of a yellowish-brown color with a reddish-brown meshwork. The kidneys each weigh 190 grams. Their capsules are adherent, the surface granular, and on section are reddish-brown in color; the cortex and medulla are not sharply differentiated and an old infarct is present on the right side. The stomach is dilated and the mucosa injected. The endometrium is thickened and congested and small ulcers are seen in the cervix.

The whole left leg and thigh are edematous. Commencing in the left iliac vein near its junction with the right is a recent soft hemorrhagic thrombus, extending down the external iliac and femoral veins and their tributaries, being especially noticeable in the saphenous and profunda veins.

May F., aged seventeen, admitted August 27, 1923, with swelling of the feet, abdomen, and back, shortness of breath, and cough.

She suffered from measles, whooping-cough, chickenpox, and mumps in childhood. She also had quinsy last March, but has not had rheumatism. The menses began in March and stopped after the second appearance, when they were profuse, with large clots, and lasted two weeks.

As a child she never played games or took much exercise owing to weakness and shortness of breath. She was excused from gymnasium on account of a heart murmur.

The attack of quinsy lasted two weeks, and was lanced by her doctor. In June the feet began to swell, and this extended up to the thighs and abdomen. Her nights were disturbed by cough, she became short of breath even at rest, and had occasional nosebleeds.

On examination she is an undersized, poorly nourished girl. The face is pale and has a muddy tinge. The lips are cyanosed. There is edema of the feet, legs, abdominal and thoracic walls, and right hand. The pulse is 120, small, regular, blood-pressure 120/96. The cardiac impulse is in the fifth space 9 cm. from the midline. At the apex there is a well-marked thrill, relative cardiac dulness 9 cm. to the right and 12 to the left. The first sound is loud and sharp, and there is a rough presystolic murmur at the apex, running through the diastolic interval with a gallop rhythm. The pulmonary second sound is markedly accentuated. At the right base there is dulness from the third spine down, diminished expansion over this side, absence of breath sounds and vocal fremitus over the dull area.

The tonsils have been removed, a portion of the left imperfectly. The liver border is felt for 5 cm. below the costal border and the vertical dulness measures 18 cm. There is movable dulness in the abdomen, indicating the presence of fluid. The urine is acid, 1024, 1.5 grams of albumin to the liter (Esbach), pus-cells, hyaline and granular casts. The quantity is 300 to 400 c.c. daily.

The electrocardiogram shows a right preponderance.

The progress of the case may be summarized as follows: Shortly after admission she was given Guy's pill—squills, calomel, and digitalis āā 1 grain. After taking 36 grains of digitalis (about equal to 4½ drams of the tincture) free diuresis set in, 1650 to 2350 c.c. of urine being passed daily, with marked diminution of dropsy and alleviation of dyspnea. She lost 10 pounds in three days. There was, however, no material fall in the pulse-rate, which continued from 100 to 120, small, but always regular. Unfortunately, a period of fever set in on September 14th to October 21st, lasting five weeks, temperatures of 101° to 102° F. being reached daily. The urinary secretion fell to 400 to 500 c.c. daily and dropsy increased. The fever was attributed to a tonsillar infection, causing much pain and accompanied by redness of the throat and sloughing of the left tonsil, which had been only partly removed. Repeated blood-cultures made during this period were negative, and there was no splenic enlargement, these features militating against the probability of fever being due to a subacute bacterial endocarditis. Fluid collected in the right pleura and was withdrawn on two occasions, September 4th and 28th, 1800 and 1500 c.c. being removed, with much relief to the breathing. A coarse pleuritic friction was heard after aspiration. Orthopnea and periods of dyspnea continued during her stay in hospital, with edema of the legs and effusions in the serous sacs. She was tormented by a frequent hacking cough and had occasional vomiting. On January 14th, a month before the termination of life, a positive venous pulse developed in the neck, attributed to relative tricuspid regurgitation. The liver increased in size and its lower border was felt midway between the umbilicus and pubes; pulsation was never noticed, but there was a light grade of jaundice.

On January 29th she complained of pain in the left side of the neck on turning the head. The left external jugular vein formed a tender cord from the jaw to the clavicle and the subclavian vein could also be felt on each side of it just behind the clavicle as a tender indurated cord. Edema was present about the elbow, which increased, and was most marked about the

elbow, giving a spindle shape to the arm, and there was also considerable edema of the hand. The infraclavicular surface veins were prominent and distended. She gradually sank and died on February 15th.

Abstract of autopsy by Dr. Parlow: There is general anasarca with effusions in the pleuræ, pericardium, and peritoneum. The heart weighs 350 grams and is enlarged, soft, and flabby. All chambers of the heart are enlarged, especially the left auricle and right side. There is slight thickening of the aortic valve due to small brownish vegetations. The mitral valve is much narrowed, semicircular, measuring $\frac{1}{2}$ by $\frac{1}{4}$ inch. The cusps are fused, but are translucent and not thickened, and there are firm pinhead-sized vegetations near its free border on the auricular side.

There is a small infarct in the left lung and adhesions at the back of both pleuræ.

The liver weighs 2150 grams; it is smooth, much enlarged, and soft. Its cut surface has a nutmeg aspect and the vascular channels are greatly enlarged. The kidneys, weighing 120 and 140 grams, show venous congestion.

Commencing in the innominate vein and extending 2 inches up the internal and along the external jugular and subclavian veins is a firm adherent thrombus, partially organized and composed of fibrous tissue, with blood-clot in its meshes, the oldest portion being at the jugular bulb. Microscopically organization and canalization is found, with a network of capillary vessels involving the outer part of the thrombus and extending from the vessel wall.

In this patient the history of shortness of breath and inability to join in games at school dates from childhood, and, unlike the first case, gave no history of acute rheumatism. The history of quinsy and the severe sore throat in hospital suggest a possible source of infection, but the symptoms of cardiac disease preceded the tonsillar attacks of which we obtained evidence. Chronic infection of the tonsil is, however, often passed unnoticed, and may have existed long before the quinsy of last March. About one-third of the cases of mitral stenosis give no

history of acute rheumatism, but in children the rheumatic or some allied infection frequently attacks the heart, while the joints escape or are so slightly affected as to escape the notice of parents who are not particularly observant.

Contrasted with the first patient dropsy was a much more prominant feature and involved the serous sacs. The liver particularly was enormously enlarged and, unlike the first case, the passive congestion was not relieved by free menstrual hemorrhage. The development of fever was followed in two days by cessation of menstruation and by an increase in dropsy and dyspnea, doubtless due to an injurious effect of an infection on the myocardium.

The feature to which I wish to particularly direct your attention was the thrombosis of veins on the left side of the neck. Pain in the neck, not of a severe character, felt on movement of the head, first directed attention to the condition. The external jugular and the subclavian veins could then be made out as firm cords, only slightly tender, with edema of the left arm and hand, increasing for several days, forming a clear picture of venous thrombosis.

Thrombosis is a complication which is not often seen in cardiac disease. Miss (now Dr.) Blampin was, however, able to collect 5 instances from the records of the Montreal General Hospital and 3 from those of the Royal Victoria Hospital during a period of twenty-two years. Dr. Welch, who has dealt fully with the subject of thrombosis in cardiac disease, points out that it occurs most frequently in the veins of the neck, arm, and chest of the left side. Occasionally the thrombus extends to the right side, but this side is very seldom affected alone. The localization thus forms a striking contrast to thrombosis in general, which affects the lower extremities in the great majority of instances.

It is particularly in disease of the mitral valve that this localization of thrombosis is observed. In 21 out of 24 of Dr. Welch's cases, 6 had stenosis, 6 stenosis and incompetence, and 9 incompetence of this valve, and in 9 Montreal cases Miss Blampin found the mitral valve affected in 7, in 1 the aortic

only, and in 1 no valvular lesion. We may, therefore, regard this form of thrombosis as particularly associated with mitral disease.

The formation of thrombus in the upper part of the body indicates that special influences are at work, and these are to be found in certain mechanical factors. It occurs in advanced stages of cardiac disease when dyspnea, dropsy, and cyanosis are usually present, conditions which favor the slowing of the circulation from venous stasis. The oblique course of the left innominate vein also tends to slow the blood-current on this side, while additional influences are the pressure exerted by the dilated left auricle and pulmonary vessels. Dr. Welch also attributes some importance to a whirling movement of the blood appearing where currents of blood from the internal jugular and subclavian veins meet at an oblique and right angle, with varying pressures and velocities, and so explains the fact that the oldest portion of the thrombus is commonly situated in the internal jugular vein at the bulb, where the enlarged end of this vessel and the presence of valves also favor the whirling movement.

The rôle of infection in producing thrombosis is one on which much difference of opinion exists. The mere finding of bacteria in isolated cases does not prove them to be the cause, as they may represent a secondary invasion. In the cases of cardiac thrombosis bacteria, mostly streptococci, have been found in very few instances, possibly because cultures have not often been made. Streptococci were present in one of Dr. Welch's cases, and in 7 cases collected by Dr. Blampin streptococci were present in 2 and staphylococci in 1.

While mechanical causes favoring thrombosis in cardiac disease are sufficiently clear, the evidence of bacterial infection is at present inconclusive. The absence of acute endocarditis in the majority of cases and the rarity of thrombosis in chronic bacterial endocarditis may be used as arguments against bacterial infection.

In contrasting the 2 cases the question may be asked Why did one live to the age of forty-one and the other die at seventeen?

In both instances the cardiac disease developed in childhood, and a well-marked stenosis in a young child frequently leads to a fatal issue about the period of puberty or early adult life. The answer may perhaps be partly found in the fact that the first case had prolonged rest in bed up to the time of puberty, and that she suffered from no infections until the attack of tonsillitis seven weeks before admission to hospital. The second case received no special care. She attended school and possibly her myocardium suffered from a focal infection in the throat, which culminated in an attack of quinsy. It is also noteworthy that in the first case three valves were seriously damaged, while in the second the mitral valve alone was affected, the aortic being so slightly thickened as not to interfere with its function. The myocardium plays a far more important part in determining the duration of the disease than does the valvular lesion. It is, therefore, our duty to carefully guard cardiac patients from any infection which may impair the cardiac muscle, and by the early and judicious removal of focal infections, such as are so common in the tonsil in early life, to spare the myocardium from injurious influences.

CLINIC OF DRS. A. H. GORDON AND C. R. BOURNE

MONTREAL GENERAL HOSPITAL

SUPPURATIVE PYLEPHLEBITIS WITH PNEUMOCOCCUS SEPTICEMIA

THE little girl of nine years whose history is before us had missed little in the way of the infections of childhood. Among these were measles, followed by pneumonia at three, and pneumonia again at five, followed by a pleurisy with effusion.

She had whooping-cough at seven and a left-sided inguinal adenitis on two occasions, which was incised, and discharged for a long time. She was never robust and suffered from repeated "colds" and bronchitis until about six months before her present illness. She had long lain under the suspicion of tuberculosis, but that suspicion had never been confirmed.

Six months before the final illness she had a sharp attack of pain in the lower abdomen, lasting a few hours, without vomiting or fever, but since then had had remarkably good health.

Her last illness commenced suddenly on October 20th with (1) chills, (2) nausea and vomiting, and (3) slight fever. The fever was remittent, reaching 102° F. at night, and some epigastric pain developed, with a palpable spleen and slight resistance of the right abdomen, and some distention.

Examination of the heart, lungs, and central nervous system showed no organic disease.

There was no headache and no eruption, and the throat was negative. A Widal reaction was negative and the leukocyte count was 13,000.

The pain was very variable—at one time she would roll about and scream, and a little later would be placid and comfortable.

The temperature rose to an evening level of 104° to 105° F., and the pulse became very rapid and small, the vomiting frequent and distressing, and the child became much emaciated. Her nervous condition at times bordered on acute mania, and, from being mild and tractable, she became most difficult to control, and her screams, even when not in pain, disturbed the whole corridor.

Her conjunctivæ showed a faint subicteroid tint, but no bile was found in the urine.

Leukocyte counts ranged from 13,000 to 24,000, with an average of 80 per cent. polymorphonuclears. A blood-culture made ten days after the onset gave no growth, another three weeks from the onset showed a pure culture of Pneumococcus Type II.

The blood urea nitrogen on November 1st was 20 mg. per 100 c.c., and the urine showed a trace of albumin with a few hyaline casts.

The frequent complaint of abdominal pain sometimes in the upper right quadrant and sometimes in the lower was associated with resistance of the right side of the abdomen, alternating with periods of complete flaccidity. Rectal examination showed a small firm mass at the tip of the examining finger toward the right. There was no diarrhea at any time.

Lumbar puncture showed a normal fluid and the ocular fundi were normal.

An exploration of the abdomen was done by Dr. Barlow on November 8th, and showed no evidence of tubercle and no peritonitis. A band of omentum was adherent to the mesial aspect of the cecum, and at the site of adhesion there was an area the size of a 50-cent piece, which was thickened and edematous. The band of omentum was ligated and the edema was found to extend down the inner side of the cecum and along the appendix, which was removed, but showed no evidence of acute appendicitis.

Several large soft glands were found in the mesentery of the cecum. The pelvis was negative, the liver was smooth and

came down 1 inch below the costal margin, and the gall-bladder appeared normal.

No attempt was made to deal with the cecum, as the child's condition became desperate and she left the table practically pulseless.

The pathologic report upon the appendix removed was "chronic appendicitis."

The laparotomy wound broke down after a few days, discharging foul-smelling greenish-yellow pus.

Incontinence of urine developed, and on November 19th several ounces of bloody fluid were vomited. On November 20th she developed a right-sided otitis media, which discharged. The pus showed Gram-positive cocci in groups and lanceolate Gram-positive diplococci.

Frequent glucose salines intravenously and several blood transfusions were of only transient benefit, and she died from exhaustion on November 26th, six weeks after the onset.

Summary:

(*a*) Sudden onset with chills.
(*b*) Paroxysmal abdominal pain.
(*c*) Irregular, but high temperature, with rapid pulse.
(*d*) Marked nausea and vomiting.
(*e*) Variable tenderness and resistance, often in the upper right quadrant of the abdomen.
(*f*) Marked prostration and emaciation.
(*g*) Leukocytosis.
(*h*) Blood-culture positive for Pneumococcus Type II.

Clinical diagnosis: Pneumococcus septicemia.

An autopsy done by Dr. L. J. Rhea thirty-six hours after death showed a gaping granulating wound exposing the liver and large bowel, the latter matted to the wall with greenish-yellow lymph. The sutures inturning the appendix had given way and the suture line was adherent to the pelvic wall. There was free pus in Douglas' pouch, and in the mesentery of the small bowel were three pockets of pus at the end points of the superior mesenteric artery. There was no free fluid in the lesser sac. The mesenteric lymph-nodes were only slightly enlarged.

The heart showed nothing of importance. The pleural cavity contained no free fluid and the left lung was free, while the right was adherent at the base, and in the upper lobe showed a few small areas of suppuration the size of a pinhead.

The lower lobe of the left lung on the cut surface showed many dark oozing blood-clots as large as a finger-tip. There

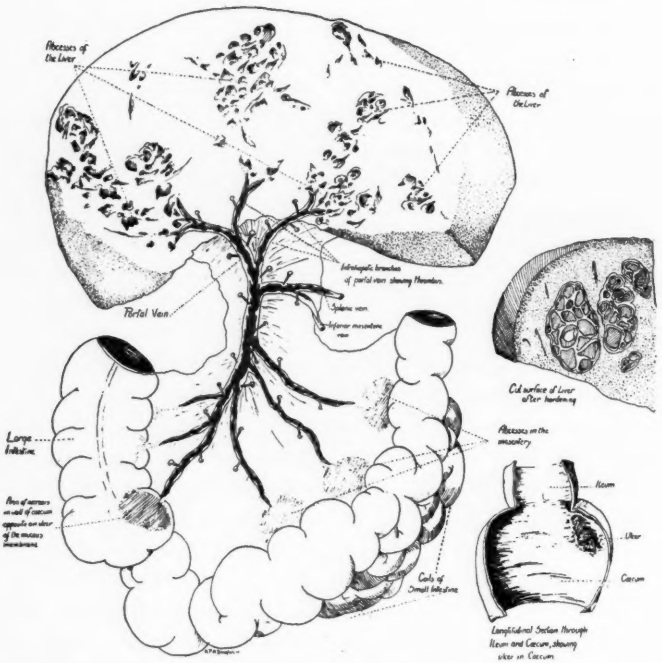

Fig. 322.—Ulcer of the cecum, thrombophlebitis of the portal vein and its tributaries, and abscesses in the mesentery. (Montreal General Hospital, A 23–247.)

was edema, but no consolidation. It was crepitant throughout.

Gastro-intestinal tract: This was negative except for an ulcer within the cecum $\frac{1}{4}$ by $\frac{1}{2}$ inch in size, situated at the region of mesenteric attachment. There was no free pus, but some

necrotic material at its base, the edges were not clear cut, the ulcerated area was soft and of a yellowish-green color.

The spleen weighed 150 grams, was dark brown, cut easily, and showed no infarcts and no abscesses.

The pancreas was normal. The kidneys showed a slight grade of acute nephritis.

The liver was very large and weighed 1640 grams. The surface was smooth and undulating. There were many irregular

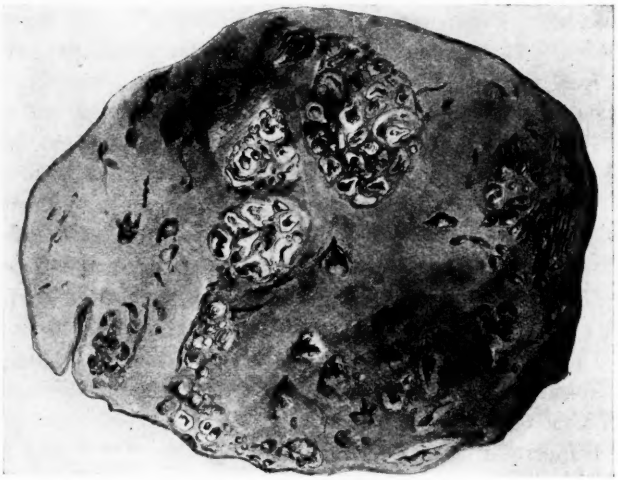

Fig. 323.—Suppurative thrombophlebitis of the portal vein, showing multiple abscesses occurring in groups.

bluish-green mottled areas upon its surface from the size of a pea to a chicken's egg. On section, a thick greenish pus exuded.

The cut surface was soft, exposing very numerous areas of abscess, as above described, and the liver substance about them was lighter than normal. Upon opening the portal vein it was found to be filled with a similar purulent substance, but of a clay color.

There was thrombosis and ulceration throughout its length, and similarly throughout the superior mesenteric vein, and its

many branches, especially those to the abscess areas of the mesentery and the area of ulceration in the cecum.

The gall-bladder was normal.

The aorta and coronary vessels were normal.

Histologic examination:

Lungs showed congestion with multiple areas of necrosis.

Spleen: Multiple foci of inflammation and some productive splenitis.

Heart: Myocardial degeneration.

Kidney: Degenerative and exudative nephritis.

Liver: Parenchymatous degeneration, fatty changes, multiple focal necrosis.

Bacteriologic examination:

The heart's blood gave a pure culture of Pneumococcus Type II.

The ulcer of the cecum showed pneumococcus and Bacillus coli.

Abscesses of liver: Bacillus coli and pneumococci.

The abscesses in the mesentery: Pneumococcus and Bacillus coli.

The spleen: Pneumococci.

Discussion.—We are confronted here with one certainty, and after that with several possibilities.

The certainty is that this child suffered from and died of a pylephlebitis—a suppurative thrombosis of the portal vein, and resulting multiple abscesses of the liver.

The possibilities are: (1) that these events were due to a pneumococcus infection, (2) that the principal disease was a pylephlebitis, not of pneumococcus origin, but that the pneumococcus was a secondary invader.

The presumable course of events in this case was infection of radicles of the portal vein from the ulcer of the cecum with upward extension of the infection and resultant thrombosis until the main trunk of the vein was reached. In addition, there was retrograde extension along the other branches of the portal vein. Reaching the liver, the thrombosis invaded the lobular veins and multiple abscesses were formed.

The flora of these abscesses were pneumococcus and Bacillus coli. (The autopsy was done thirty-six hours after death.)

It is reasonable to assume that the organism found in the blood during life (Pneumococcus Type II) was the cause of the pylephlebitis and liver abscesses, and that its primary focus was the ulcer in the cecum.

We may recall that the patient had two definite attacks of pneumonia some years before, as well as numerous "colds," and that she constantly gave the impression of laboring under some chronic infection.

Prolonged, persistent, and atypical pneumococcus infections are recognized.

Turretini[3] reports an instance in which pneumococci were found in the blood six months after the original pulmonary infection, and during that period there had been constant fever.

Bazy[7] mentions a subdeltoid hygroma infected by the pneumococcus three weeks after pneumonia, and also describes a perianal abscess containing pneumococci in a patient who had pneumonia twice, ten and twelve years before.

Lumsden[15] notes a pneumococcus septicemia with endocarditis of six weeks' duration without evidence of lung lesion.

If this case should be a pneumococcus infection of the portal vein and liver, originating from a cecal ulcer, it would appear to be unique.

There are, however, two possible weak links in the evidence.

The pneumococcus could not be obtained in pure culture from the liver abscesses on account of overgrowth of other organisms; second, the blood-culture taken on the tenth day was negative, but that on the twenty-first day was positive. This objection loses some of its force in the absence of an intervening pneumonia, which might have been a reason for a secondary blood infection.

Suppurative pylephlebitis is not a common disease, and I regret to say that when it does exist, it is frequently not recognized during life.

It would also appear from a review of the literature that

it is perhaps less common than it was a generation ago, and this may have to do with the earlier recognition and surgical treatment of appendicitis and other intra-abdominal infections.

In any event, the descriptions given by the English clinicians of twenty years ago, Langdon Brown[13] and Bryant,[1] have not been improved upon in recent years.

In the matter of etiology the following table from Langdon Brown's article gives the source in 64 cases:

	Cases.
Appendix and cecum	27
Stomach	6
Rectum	6
Duodenum	2
Dysenteric ulcer of colon	1
Typhoid fever	2
Acute enteritis	1
Total Gastro-intestinal lesions	45
Gall-bladder	4
Abscess behind pancreas	1
Suppurating mesenteric glands	1
Bristle in mesenteric vein	1
Pyemic abscess of spleen	1
Abscess of ovary	1
Empyema	2
Subphrenic and cerebral abscess	1
No associated suppuration	7
Total	64

White[12] reports a case of multiple abscesses of the liver in an infant of sixteen days from infection along the umbilical vein where the umbilicus had apparently healed perfectly.

Frequency.—Out of 9494 medical necropsies in thirty-two years at St. Bartholomew's Hospital there were 65 cases of abscess of the liver, and of these, 18 per cent. were associated with suppurative portal phlebitis, giving a total for the latter of 0.12 per cent. of all autopsies. From 1918 to 1924 there were 1643 autopsies in the Pathological Department of the Montreal General Hospital. Among these were 3 cases of thrombosis of the portal vein, of which one was a case of suppurative pylephlebitis, or 0.06 per cent.

Age.—Eighty-five per cent. occurred under forty and 60 per cent. under thirty years of age.

For a disease which is so dependent upon infection in the portal area, the rarity of portal phlebitis in typhoid and in intestinal tuberculosis is striking. Lannois,[16] however, has reported a case in which pylephlebitis with multiple abscess of the liver developed late in the course of typhoid, and typhoid bacilli were found in the pus from the liver abscess.

In this connection it is of interest that in the great majority of cases pylephlebitis has been associated with suppuration outside of the lumen of the bowel.

Duration.—The average duration was forty-four days, though Bryant[1] quotes one case which lasted two hundred and ninety-six days.

Associated Suppuration.—Frerichs found this to be rare—4 cases out of 25—but Langdon Brown's[13] statistics showed that about 50 per cent. showed peritonitis, and, in addition, 33 per cent. showed respiratory infections, 10 per cent. cerebral abscess, 3 per cent. ear suppuration, and 6 per cent. infections of kidney and of pericardium.

Symptoms.—*Pain* is almost always present and occurs early. *Tenderness* over the liver is usual. *Enlargement of the liver* occurs in 75 per cent. of cases, splenic enlargement in 25 per cent.

Jaundice occurs in about 40 per cent., is not marked, and bile is found in the stools.

Vomiting is very common and diarrhea was noted in 40 per cent. of cases.

Chills were present in 65 per cent. of collected cases.

Fever is invariable, and leukocytosis is nearly always found ranging from 15,000 to 20,000.

Delirium and *restlessness* are very common.

Prognosis is practically always fatal, although Langdon Brown cites some cases of postmortem evidence of previous healed lesions, and Moschcowitz[14] quotes a case of Gerster's which developed symptoms of pylephlebitis following appendicectomy and finally recovered. She died four years later of

hematemesis, and at autopsy the evidences were found of old healed suppurative foci in the liver.

Schiff and Claude Bernard, after ligation of the portal vein in animals, found that the animals developed a syndrome consisting of prostration, coma, nervous twitchings, and fall of temperature; and Goyet[2] describes a case of pylephlebitis with portal obstruction, occurring in the course of cancer of the stomach, in which there appeared mild delirium, somnolence, crying and moaning, and hypothermia.

He makes the suggestion that such cases may simulate the condition found in portal ligation and Eck's fistula.

Neuhof[9] found that portal ligation in dogs caused death in a short time, with rapid irregular pulse and respiration, coma, absent reflexes, and paresis of the hind limbs.

On incomplete ligation a collateral circulation developed, and later a complete ligation did not cause death. He therefore suggests the ligation of the portal vein in pylephlebitis, since collateral circulation will then have occurred, and the vein may be excluded, as is the jugular in sinus thrombosis, and he suggests that thus the liver may be able to take care of the infection.

Beer[10] carried out this procedure on a patient after having done an omentopexy previously, and though the patient succumbed, at autopsy there was no evidence of portal obstruction.

In a disease as hopeless as the one under consideration one might seriously consider such a procedure, as undoubtedly all other treatment has up to the present been symptomatic only.

(We wish to acknowledge with thanks the assistance of Miss H. Douglas in the preparation of drawings of the pathologic specimens.)

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CLINIC OF DR. D. S. LEWIS

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SYPHILIS OF THE INTESTINE

GENTLEMEN: I should like to present for your consideration 2 cases in whose development syphilis has played an unusual rôle. That syphilis is a general infection in which every system of the body suffers has been recognized since the time of Lancisi and Morgagni, and its manifestations are of such protean character that Osler was in the habit of telling his students that to know syphilis was to know medicine. It is, therefore, difficult to understand why syphilitic lesions of the digestive tract should have been almost ignored until the beginning of the present century. However, the discovery of the Treponema pallidum by Schaudinn in 1905, and of the Wassermann test in 1906, gave the diagnosis of visceral syphilis a great impetus, and we are now being forced to recognize the comparative frequency of syphilis of the digestive tract. Such diagnoses previously had been made chiefly at autopsy or as accidental findings at operation (Elder), but now are becoming more frequent at the bedside.

A review of the more recent literature shows that the infection attacks certain regions of the alimentary tract more frequently than others. Syphilis of the esophagus and of the small intestine is rare, while a rectal lesion is relatively the most common. Symmers, in 4880 autopsies, found gastric syphilis once, as compared with 6 cases of syphilis of the intestine, 4 of the latter being in the rectum. But, on the other hand, Wile and Mills both believe syphilis of the stomach and of the intestine to be almost equally frequent in their occurrence. During the

past few years, however, a considerable group of cases of gastric syphilis have been described, interest in the subject having been stimulated by the similarity of the *x*-ray picture to that shown by cancer of the stomach. In other words, the importance of differentiating the two conditions has aroused a very active study which has done much to clarify the diagnostic picture. Similar work is required in the clinical study of luetic enteritis.

Syphilis of the intestine may be congenital or acquired, and the site of the lesion varies according to the particular type present. In congenital syphilis the lesions are most usually in the ileum, while in the acquired type of syphilitic enteritis the lower part of the colon and rectum are involved, but MacCallum also reports acquired tertiary lesions in the upper ileum and jejunum. The distribution of the acquired syphilis differs materially from that seen in tuberculous enteritis, where the site of election is the lower ileum and cecum, while the upper and lower parts of the tract usually escape.

The secondary rash may quite possibly affect the intestinal mucosa simultaneously with the manifestations in the mouth and skin. The symptoms should be those of an acute catarrhal enteritis, with mild abdominal pain and diarrhea, but these cases respond so speedily to treatment that no reference need be made to this stage of the acquired disease. The lesions usually met with are those of a tertiary nature. They appear as low elevations of the mucosa and submucosa, which later break down to groups of ulcers which encircle the gut, and which on healing give rise to cicatrix formation and stenosis of the lumen. The microscopic picture is that of any granulomatous inflammation—a marked round-celled infiltration, chiefly perivascular in distribution, endarteritis, and, according to the stage of the process, the fibrotic side of the picture will be of greater or less prominence. Amyloid degeneration may arise, but is not necessarily present.

From this description it will be seen how easily syphilitic may be confused with tuberculous lesions, both in the ulcerative and sclerotic stages, and it is quite possible that certain

cases diagnosed as syphilis of the intestine may, in reality, be tuberculous in origin, or vice versa, and the differentiation may be extraordinarily difficult even on histologic examination, as will be seen in the second case today.

The symptoms of syphilitic enteritis vary with the type and location of the disease. In the early ulcerative stage there will be the signs of chronic enteritis: pain, diarrhea alternating with constipation, the presence of blood and pus in the stools. In the sclerotic and obstructive stage there will be signs of obstruction, and if the lesion be in the lower bowel, a malignant tumor of the rectum may be simulated with ribbon stools and tenesmus. Perforation is not unknown.

With this description of the disease in mind, I should like to present the following cases:

CASE I

The patient is a married woman, thirty-eight years of age.

Complaints.—Diarrhea; pain in back and rectum; frequency of urination; increasing weakness and loss of weight.

Personal History.—With the exception of a sore throat in 1920, she was in good health until the onset of present illness. She has been married fifteen years, but has never been pregnant, her periods being regular until December, 1921, since when they have been absent.

Family History.—Not of interest.

Present Illness.—The onset dates from December, 1921, when she noticed a gradually increasing pain in the right loin. At the same time a persistent diarrhea made its appearance. There were from eight to ten very foul, watery motions daily, these were followed by much tenesmus, and at times the stools contained blood. Associated with this diarrhea there was a marked loss of weight (about 25 pounds) and an increasing weakness, so that she could no longer do her housework. There was occasional double vision and a marked loss of memory. The latter makes the finer points of the history unreliable. She has never had any cough or night-sweats, and she has had no headache.

Physical Examination.—Temperature, 98° F.; pulse, 80; respirations, 18. The patient is a small, emaciated female, distinctly pale, and the skin is of a dark color and very dry to the touch. The pupils are irregular in size and react sluggishly to light. The teeth are artificial, the tongue smooth, and the tonsils atrophic. There are no scars about the palate. The glands generally are palpably enlarged, and those in the inguinal and femoral groups are the size of peas and shotty.

The bones of the lower extremities show a marked rachitic deformity.

The respiratory movements are equal on both sides of the chest; the percussion note is hyperresonant, and the breath sounds are well heard and vesicular in character; no râles can be made out. The heart is not enlarged, the sounds are well heard, regular, and are not accentuated. The vessel walls are just palpable; the blood-pressure is 110/75.

The abdomen is distinctly full in the lower half, and shows very evident peristaltic waves in both lower quadrants. The liver edge is sharp and extends from the ninth rib on the left costal margin down and across to the iliac crest in the right flank. The surface is quite smooth and the organ is not tender. The spleen cannot be felt. There is some fulness and tenderness in the left lower quadrant of the abdomen, but no definite mass can be made out. Rectal examination shows no external hemorrhoids, but about 2.5 cm. above the anus the walls of the rectum become rough and granular, and at 7.5 cm. above the anal margin one comes to a stricture which just admits the tip of the finger. The walls of the rectum are very inelastic, but are not greatly thickened. The entire circumference of the rectum seems to be involved and does not suggest new growth, but rather something of inflammatory origin.

Pelvic examination shows a much thickened urethra, but otherwise healthy vagina. The parametrium is greatly thickened, related in part to the rectal condition. The uterus is not unduly large, and the appendages are not felt.

The cranial nerves are normal except for the irregularly shaped and sluggish pupils. The patient complains at times of

double vision, but no definite ocular muscle paralysis can be made out.

The knee-jerks and ankle-jerks are present and equal. On the left there is a dorsal flexion of the toes, while the plantar response is normal on the right. The superficial abdominal and epigastric reflexes are active.

Urine.—Acid, turbid, specific gravity 1024, contains a trace of albumin. Microscopic examination shows epithelial débris, many leukocytes, and occasional granular and hyaline casts.

Stools are liquid, dark brown, and very fetid. They contain many pus-cells and give a strong benzidin reaction.

Blood.—Wassermann, ++++; R. B. C., 3,190,000; W. B. C., 10,800; Hgb., 75 per cent. (Sahli); the differential formula is normal.

Cerebrospinal Fluid.—Clear and under normal pressure, the globulin content is increased, there are 34 cells per cubic millimeter, mostly lymphocytes. Wassermann, ++++.

Special Examinations.—The proctoscope confirms digital examination. The instrument could not be passed through the stricture.

***x*-Ray Studies.**—Barium meal shows a normal stomach, and a rapid passage of the barium through the intestine (diarrhea). A barium enema shows a marked stenosis of the rectum and sigmoid, confirming the direct examination. The enema entered in a thin stream upward, then across to the right almost horizontally (Figs. 324, 325). The barium then passed slowly back to the left and up along the descending colon. The stricture appeared about 10 to 15 cm. long and there was no great dilatation of the colon above the obstruction.

x-Rays of the long bones of the legs showed a marked bowing, but no evidence of syphilitic periostitis.

Progress of Case.—Since her admission the patient's temperature has usually been below 99° F., but occasionally it has reached 100° or 101° F., and the pulse has been somewhat rapid (80-120). There have been from six to ten very fetid stools daily.

In view of the positive Wassermann in blood and spinal fluid, antisyphilitic treatment was commenced. She received

two short courses of mercury by inunction, in all fourteen rubbings were given, and novarsenobenzol (Billon) 0.3 gm. was given intravenously on November 10th, and 0.45 gm. seven days later. After the second injection the patient developed a marked toxic dermatitis, chiefly over the trunk, but without jaundice or any other sign of hepatic injury, nor was there any increased albuminuria. In view of the above, arsenic treat-

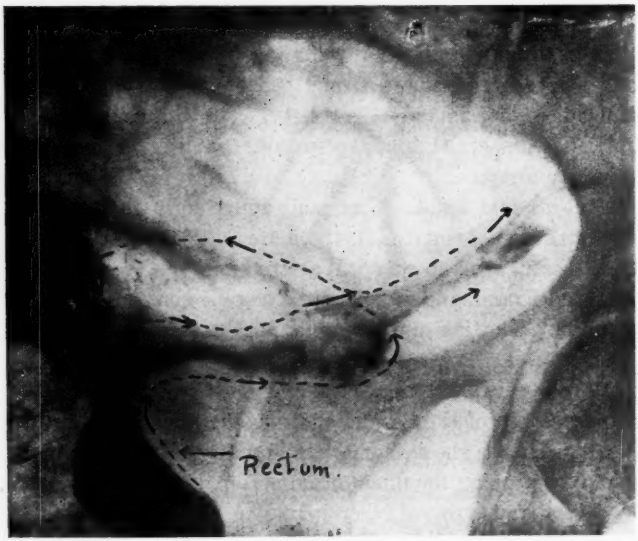

Fig. 324.—Case I. Early stages of barium injection. Markedly stenosed and tortuous lumen of rectum and sigmoid. Course taken by enema indicated by arrows and dotted line.

ment was discontinued, and the mercurials were also stopped because of signs of intolerance. The diarrhea was checked for a short time following the salvarsan, but the improvement was of a temporary character.

To recapitulate: We have in this patient a history of prolonged diarrhea with bloody and fetid stools, marked rectal tenesmus, emaciation and weakness, a positive Wassermann in blood and spinal fluid, a large smooth liver, a well-defined long

and narrow stricture of the rectum, the walls of which are extremely hard and resistant, with evidence of ulceration of the bowel, at least in the constricted area. The barium enema shows a normal-sized colon above the stricture.

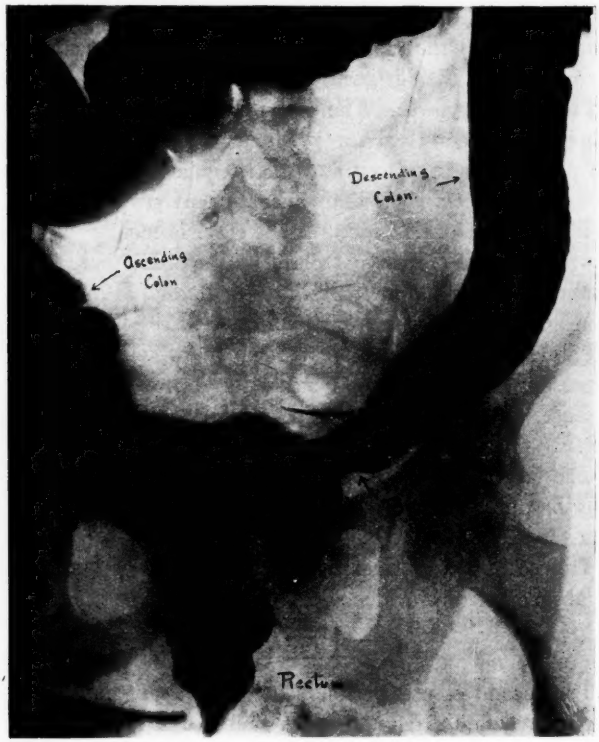

Fig. 325.—Case I. Enema injection completed. Stenosed rectum and sigmoid from *A* to *B*, above which is the undilated descending colon.

In view of the above we are justified in making the following diagnosis: (1) Syphilis; (2) syphilis of the central nervous system; (3) stricture of the rectum and chronic ulcerative proctitis (syphilitic); (4) myocardial degeneration; (5) enlarged liver (amyloid?); (6) rachitis (old).

Discussion.—In syphilis of the rectum, which is much more common in the female than in the male, the rectal and proctoscopic examinations are usually characteristic. While primary and secondary lesions occur, they are relatively unimportant as compared with those of the tertiary stage. In syphilitic stricture the great feature is the length of the stenosed area. To the examining finger the stricture is more rigid and inelastic than in carcinoma, and the surface, while ulcerated, lacks the friability of a malignant growth. The whole circumference of the rectum is involved rather than the predominating unilateral distribution of the mass in cancer, and the irregularity of surface is less noticeable in rectal lues than in malignant growths.

The barium enema picture, however, is most typical, as is shown in the accompanying plate. In syphilis there is a long, small channel fading gradually into the undilated hypertrophied colon above. This is in sharp contrast to the localized irregularity of the filling defect of carcinoma, which, if obstruction be present, is followed by a markedly dilated colon above the stricture, an expression, as Mills remarks, of the more sudden onset of the stricture.

The large, smooth liver is not tender and has none of that stony hardness which characterizes the organ riddled with small metastases.

Tuberculosis as a causative agent may also be excluded by the absence of any evidence of systemic tuberculosis.[1]

CASE II

The second case is presented as evidencing the difficulty of making a definite diagnosis of syphilis of the small intestine, even with the assistance of an anatomic examination. The presence of a positive Wassermann and symptoms of obstruction which disappeared after treatment cannot be taken as definitely proving the presence of a syphilitic lesion, although such would be a natural inference.

[1] The patient died one month later. The necropsy confirmed the clinical diagnosis as regards the stricture and ulcerative proctitis. The liver, however, showed a marked fatty degeneration and nothing more.

The patient again is a female, aged fifty, and a cook by profession.

Complaints.—Pain in the left side of the abdomen; vomiting, loss of weight.

Present Illness.—Onset three years ago with severe cramp-like pain referred to the umbilical region, and occurring most frequently at night. The pain was not influenced by the type or amount of food eaten during the preceding day, and it was described as severe, throbbing, accompanied by much rumbling of gas, and it occasionally radiated through to the back. It would often last five or six hours, and during that time much wind would be brought up, but none passed per rectum. Food, baking soda, etc., gave no relief. The spasm of pain was usually followed by the vomiting of large amounts of watery and very sour material, of which the latter part was often very foul in odor and of a brownish color, but no actual blood was ever noticed in the vomitus or stools. The vomiting always gave relief. These attacks have occurred once or twice a week, and of late the pain has become much more severe, and at the height of the attack she has noticed movements over the upper part of the abdomen, which promptly disappeared with the relief of the pain. Fear of the pain has prevented her eating at all freely, and her weight has fallen from 180 pounds six years ago to 104 pounds at present.

Personal History.—She has had measles, rheumatic fever, tonsillitis, and hemorrhoids. The menopause occurred five years ago, and there has been no subsequent blood loss. She has had 4 children and no miscarriages. Her husband died of alcoholism and is reputed to have infected the patient with "venereal disease" some years before his death.

Family History.—Her father died of tuberculosis, as also did one of her own children. Otherwise family history is negative.

Physical Examination.—The temperature and pulse-rate are normal. The patient is an emaciated female who looks rather older than her stated age (fifty). The teeth are artificial, the tonsils small, and tongue pale. The pupils are equal and react

to light. The chest shows a little lagging on the left side, with a slightly higher pitch to the percussion note over the left apex. Beyond a few moist râles at the bases, and some doubtful changes in the respiratory murmur over the right middle lobe, nothing unusual is to be made out over the lungs. The heart is slightly enlarged, the sounds are regular and well heard, with an increased sharpness in the second sounds at the base. There is a soft systolic murmur over the whole pericardium, but this is not transmitted to the axilla. The peripheral vessels are somewhat thickened and the blood-pressure is 134/64. The abdomen is rounded, the lower half showing a distinct fulness, while from time to time definite visible peristalsis may be seen in the left upper quadrant. There is a vague sense of resistance just above the umbilicus, and a diffuse tenderness over the lower quadrants. Rectal and pelvic examination reveal nothing of interest. The reflexes are active.

Urine.—Acid, cloudy, specific gravity 1013; no albumin and no sugar. Microscopic examination shows many white blood-cells and much epithelial débris. Bacteria are also present in large numbers.

Stools.—Contain no blood and are negative as to parasites.

Blood.—Wassermann, ++++; R. B. C., 3,300,000; W. B. C., 3500; Hgb., 68 per cent. (Sahli). Differential count is normal, and there is no anisocytosis or poikilocytosis.

Special Examinations.—*Gastric Analysis.*—Following a test breakfast of 30 gm. of bread and 400 c.c. of weak tea no free acid could be found, while the total acid varied between 6 and 12 per cent., with an occasional trace of occult blood in the different specimens.

Barium Series.—After eight hours the barium is in the small gut and appearance suggests obstruction of the small intestine. The stomach appears normal. At a second examination, one week later, the barium, eight hours after the meal, is seen in a ball-like mass in the left upper quadrant, and from this mass the barium can be seen spurting in an upward direction, evidently through a narrow part of the small intestine. In two minutes a "ladder column" of barium was seen (Fig. 326).

x-Ray Findings.—Obstruction in small intestine on left side of abdomen, probably tuberculosis or cancer, but might be due to band.

Progress of Case.—During first two weeks in hospital the pain recurred at intervals. On a liquid diet it was less severe

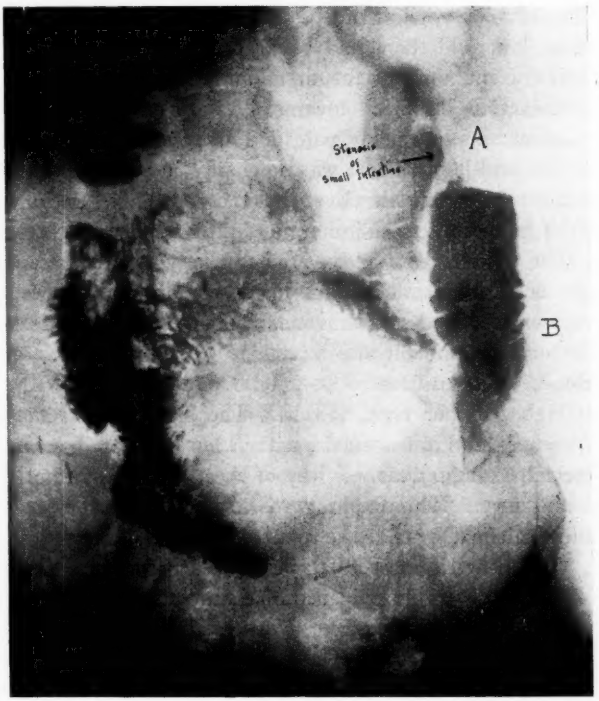

Fig. 326.—Case II. Stricture of small intestine. Barium collected behind the stricture *A*, and below is a well-defined ladder pattern at *B*. The barium can be seen passing through as a very thin band upward.

than usual, but after a single dose of arsenobenzol (0.45 gm.) there was a complete disappearance of the abdominal symptoms. The pain vanished and there were no further cramps, nor could the peristaltic movements be seen. Her appetite returned and she was able to eat full house diet with comfort.

She received three further injections of novarsenobenzol and was discharged much improved.

Diagnosis.—Partial obstruction of small intestine, syphilitic(?), etc.

Second Admission.—The patient returned some two months later for admission to hospital. She complained of general weakness, loss of color, swelling of legs and face, and almost continuous headache, but said there had been absolutely no return of the abdominal complaints of the first admission.

Examination showed a fever of 100° F. and a rapid pulse. The patient was extremely pale and there was definite edema of the face and legs. There was no change in the signs over the heart and chest, but the liver was now palpable two finger-breadths below the costal margin, in the right midclavicular line. The reflexes were active. The urine was clear except for a trace of albumin. The microscopic examination showed, as before, white blood-cells and many bacteria, but no casts. Stools contained no blood.

Blood.—Wassermann, +++; R. B. C., 1,800,000; W. B. C., 7000; Hgb., 40 per cent. (Sahli). The smear shows marked poikilocytosis and anisocytosis, with a few nucleated reds.

Special Examinations.—*x*-Ray of chest shows a normal-sized heart and aorta. The diaphragm is in normal position. There are a few calcified spots at the roots of the lungs and at the right base. The heart is more to the right than normal. A second barium series shows no evidence of the obstruction seen in the preceding examination.

Subsequent Progress.—There was a marked reduction in the edema, but no improvement in the anemia followed the usual remedies. The red corpuscles steadily fell to 1,200,000, with a hemoglobin of 25 per cent. There was an increasing irregularity in size and shape of the cells, and less and less sign of any regenerative activity of the bone-marrow. Two months before death a bronchitis first made its appearance; six weeks later fluid appeared in the pleural cavities. The fluid was clear, straw colored, had a specific gravity of 1015, and cells were chiefly lymphocytes. Death finally occurred, with broncho-

pneumonia and myocardial failure, seven months after her first admission.

Discussion.—The clinical diagnosis in the case is one of considerable interest. We have to deal with a twofold picture. Is the condition the exceedingly rare syphilis of the small intestine with a superimposed anemia of syphilitic origin, or are we dealing with a tuberculosis of the bowel secondary to an old pulmonary lesion?

The history and *x*-ray findings leave no doubt that on admission there was a very definite stenosis of the small intestine, probably in the upper part, but the etiologic factor cannot be settled so readily.

In favor of the more attractive diagnosis of syphilitic stenosis we have the history of venereal infection, the strongly positive Wassermann in the blood, and the startling relief from all symptoms after a single injection of novarsenobenzol. This relief was apparently permanent, and at subsequent *x*-ray examination no evidence of obstruction could be made out. The stricture, according to the therapeutic test, could be regarded, therefore, as having some relation at least to syphilis. Again the rapidly fatal anemia of the hemolytic and, later, aplastic type, associated with the positive Wassermann, suggests the presence of a fairly active syphilitic process. Syphilis of the small bowel is usually of the congenital type, but MacCallum also describes tertiary acquired syphilis in the same location.

On the other hand, tuberculosis of the bowel is relatively common, and in its hypertrophic form it does occasionally cause obstruction, with multiple stenoses, but here again the location is unusual, tuberculosis of the bowel being most generally in the neighborhood of the cecum and ileocecal valve, while in the present instance the obstruction is quite high up in the small intestine. In favor of tuberculosis we have the family history of the father and son dying of the disease, the physical signs in the chest, the *x*-ray findings of calcified nodules in the roots of the lungs, and the increased density at the right base, and again the final history of a terminal bronchitis, with a pleural effusion rich in lymphocytes.

The possibility of the obstruction being of a malignant nature can be excluded with considerable authority by the permanent disappearance of the evidences of obstruction, and also by the absence of any signs of metastases at any subsequent time.

In this instance the case came to autopsy with the clinical diagnosis: (1) Syphilis; (2) chronic partial obstruction of small intestine (syphilitic?); (3) anemia (syphilitic?); (4) pulmonary tuberculosis (old); (5) pleurisy with effusion; (6) bronchopneumonia.

Autopsy Report (Abridged)

Abdomen (Dr. Cochrane).—*Stomach:* The mucosa shows no rugæ, and there are many petechial hemorrhages.

Small intestine: About 96 cm. from duodenojejunal junction there is a definite fibrous constriction 2.5 cm. long. Above this constriction the bowel is slightly dilated and the walls thickened. Just above and below the constriction are ulcerated areas of mucosa, and the adjacent mesenteric glands are hard and shotty. At a distance of 107 cm. from first constriction is a smaller, but similar narrowing, 2.5 cm. long, and again 12.5 cm. below this there is another, while a fourth scarred ulcerated area is 48 cm. below the third. The last does not seem to have led to any stenosis.

Large intestine: Normal in appearance.

Sections of the stricture: There is a thinning of the mucosa, with thickening of the submucous and muscular coats. Scattered throughout, but occurring especially between the submucosa and muscularis and between the muscular coats and in the adventitia, are definite patches of granulomatous tissue. All stages are seen, the earliest showing small round-cell mantles, epithelioid whirls, and giant-cells. There is no caseation, but there is some perivascular infiltration and thickening. *Ulcers:* Here the gut is not thickened; the base of the ulcer only extends into the submucosa, and is formed by inflammatory tissue with a large number of small mononuclear and plasma cells with few polymorphonuclears. There is no caseation. The granulomatous inflammation is more diffuse in the submucosa, but one can distinguish similar localized areas with epithelioid and

giant-cells as described above. The picture is one of granulomatous inflammation and granulomatous ulcer.

The mesenteric glands show similar changes, also without caseation.

The pleura is markedly thickened over the right middle lobe and contains typical early and fibrous tubercles, which also spread irregularly through the lung in a lymphogenous extension.

Anatomic Diagnosis.—Ulcerative and productive stenosing enteritis; anemia (syphilitic?); tuberculosis of lungs and pleura; bronchopneumonia.

The above findings in the chest and the rather equivocal nature of the intestinal lesions prevent the pathologist making a diagnosis of syphilis of the intestine. A definite differentiation was not made, but it was generally felt that the burden of proof lay with those favoring the syphilitic origin. Aschoff states that in certain cases it is very difficult even at necropsy to differentiate between the syphilitic and tuberculous nature of the stricture. We are certainly dealing with a dual infection—tuberculosis and syphilis—but whether it is syphilis with an engrafted tuberculosis, or the reverse, is difficult to say.

Treatment.—As Mills, of St. Louis, remarks, syphilitic lesions of the bowel may be of two types: (1) Primarily luetic and so amenable to medical treatment; or (2) primarily sclerotic with motor impairment, where the treatment will necessarily be of a surgical character. In the first there are signs of a chronic and exhausting enteritis, with copious diarrhea, and here the results of medical treatment are satisfactory in the extreme. The diarrhea ceases, the patient rapidly gains strength, and with the cure of the disease there is a complete return to health. There is, of course, in these cases always a question of the accuracy of the diagnosis—as it is made on the basis of the therapeutic test and by inference only. Judged by such standards, our second case would undoubtedly be classed as a syphilis of the intestine had she not returned to the hospital at a later date.

In the second, or sclerotic group, the symptoms are largely

those of obstruction. There is a large deposit of fibrous tissue upon which medical treatment has no effect. Here the treatment must be surgical. Dilatation has proved unsatisfactory where the stenosis is accessible (rectum), and colostomy is the one means of relieving the diarrhea. Rarely is it possible to resect the stricture, and even in these cases the outlook is generally unfavorable. The first case falls in this category.

These 2 cases are of interest—the first as a relatively uncommon form of stricture of the rectum, the second as an example of the difficulties which may arise in differentiating the lesions of tuberculosis and syphilis when both infections occur in the same individual. With the increasing use of the serologic and radiologic methods of diagnosis it is to be expected that syphilis of the digestive tract will become a more frequent bedside diagnosis, and it is with a view of stimulating interest in this regard that these 2 cases are presented.

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CLINIC OF DR. S. GRAHAM ROSS

ROYAL VICTORIA HOSPITAL

PNEUMOTHORAX IN INFANCY

I WISH to present 3 cases of pneumothorax occurring in this service during the past year. The patients were all infants aged three, five, and twenty-three months respectively, and each one illustrates a separate type of this disease. All the patients showed pyopneumothorax.

Case I.—Age three months; was admitted to the Royal Victoria Hospital on January 22, 1923, with complaints of cough and loss of weight.

Family History.—Father suffering from advanced pulmonary tuberculosis. Mother in poor health, but showing no evidence of active tuberculosis. She has had 6 children, 4 of whom died between the ages of five months and three and one-half years of the following diseases: diarrhea, meningitis, diphtheria, and influenza.

Personal History.—Small child, at birth weighing 5½ pounds. Breast fed one month. Since then on proprietary foods and cow's milk dilutions. No digestive disturbances.

Present Illness.—Caught cold two weeks ago. Since then has had cough. Has lost weight and vomited occasionally.

Present Condition.—A fairly well-nourished infant, weighing 8 pounds, 7 ounces. Temperature 98° F., pulse 110, respirations 36. No superficial glandular enlargement. Head, eyes, ears, and mouth negative. The chest is symmetric. Lungs resonant throughout to percussion. Breath sounds of normal puerile character. A few large moist râles heard throughout, which clear up on coughing. Heart normal. Abdomen normal. Urine normal. Blood: W. B. C., 16,000; R. B. C., 3,600,000; H. B., 70 per cent.

Course of Disease.—An intradermal tuberculin test with $\frac{1}{10}$ mg. was done on admission. This showed a doubtful positive reaction. On January 31st the temperature rose to 100° F. Examination of ears showed left otitis media. On February

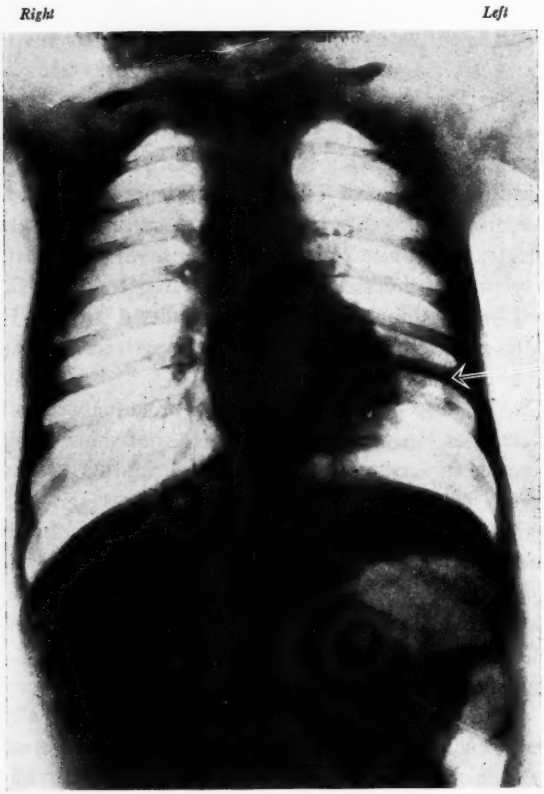

Fig. 327.—Shadow in lower part of left lung indicates a focus of tuberculosis. Arrow points to this.

1st temperature was $101\frac{3}{5}$° F. Double otitis media. Paracentesis of both ear-drums. Slight diarrhea. February 3d: Temperature normal. Child losing weight. Both ears discharging freely. February 6th: A few medium moist râles

heard in right axilla. Profuse aural discharge. February 7th: A second intradermal tuberculin test of $\frac{1}{10}$ mg. shows a definitely positive tuberculin reaction. The spleen is palpable.

x-Ray of chest (Fig. 327). Report: "Slightly increased density at root of right lung. Patch of increased density in lower part of left lung. This patch may be due to a tubercular focus."

On account of an outbreak of chickenpox in ward patient was discharged.

Diagnosis: 1. Tuberculous tracheobronchial lymph-nodes. 2. Bilateral suppurative otitis media. It was suspected that there was also a primary focus of tuberculosis in the left lung as demonstrated by the *x*-ray. Patient developed varicella ten days after discharge, from which he made a good recovery.

Second Admission.—Admitted to hospital again on April 10th, at age of five months. His cough has persisted during his stay at home, and during the past week has become worse. Weight has remained stationary.

Present Condition.—Poorly nourished and appears ill. Weight eight pounds. Temperature 97° F. Pulse 100. Respirations 30. Shows a grayish pallor, with slight cyanosis. Subcutaneous tissues scanty. No superficial glandular enlargement. Head and eyes normal. Ears: Left drum opaque, shows scar of old perforation. Right drum opaque, small perforation in posterior half. No discharge. Mouth normal. Chest symmetric. Both sides move equally on respiration. Lungs: Percussion note normal throughout. Breath sounds puerile. Over the left upper lobe and high in left axilla a few fine crepitations are heard. An occasional moist râle is heard in right lung. Heart normal. Abdomen rounded. Spleen palpable. Urine negative. Blood: W. B. C., 14,000; R. B. C., 4,100,000; H. B., 65 per cent. Wassermann on blood negative.

Course of Disease (April 10th): This evening patient had a sudden attack of acute dyspnea, with violent coughing lasting several minutes. Examination shows very slight respiratory movement of chest. On percussion left side of chest is definitely hyperresonant. The heart dulness is obliterated on the left

side and the heart apparently pushed over to the right, the apex-beat being felt just to the left of the sternum. Over left chest the breath sounds are distant and blowing. Over the left upper lobe numerous tinkling musical râles are heard. The

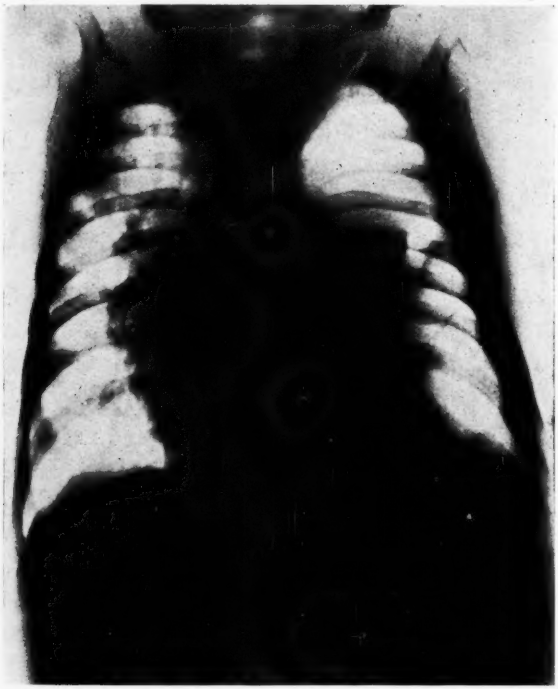

Fig. 328.—Pneumothorax of left pleural cavity. The cavity appears to be divided into two parts by adhesion of the lung to the chest wall in the region of the fifth rib. The parietal pleura is thickened. The heart and trachea are pushed over to the right.

characteristic coin sound is present. Diagnosis: Pneumothorax of left pleural cavity.

April 11th: Left side of chest shows bulging. Physical examination as on previous evening. *x*-Ray of chest shows a left-sided pneumothorax with heart displaced to right.

April 12th: Slight rigidity of neck.

April 13th: Discharge from right ear. Fine crepitations heard throughout right lung. *x*-Ray of chest (Fig. 328). Report: Left pneumothorax with adhesion of left lung to region of fifth rib at its lateral aspect. The upper part of the pneumothorax extends across to the right lung, reaching about 1 inch beyond the middle line. The trachea and heart are very markedly displaced to the right, little or no fluid present. Left pleura shows definite thickening.

April 15th: Cough is more severe. There is dulness on percussion at left base suggesting fluid. A specimen of sputum obtained with swab from throat shows presence of acid-fast bacilli. The voice sounds at lower angle of scapula are of a distinctly amphoric quality.

April 17th: Lumbar puncture attempted, but stopped on account of attack of cyanosis and cessation of breathing.

April 18th: The intradermal tuberculin test to $\frac{1}{10}$ mg. showed decided positive reaction. Dyspnea more marked. Patient died.

A puncture of cisterna magna was done immediately after death; 5 c.c. of turbid fluid was obtained. Cell count 1200, predominantly lymphocytes. No tubercle bacilli found. Smear of spinal fluid shows numerous Gram + lancet-shaped diplococci of morphology of pneumococci (capsulated). Possibly a terminal pneumococcal meningitis. Throughout stay in hospital temperature did not rise above normal.

Diagnosis.—Pulmonary tuberculosis. Tuberculous pneumothorax. Chronic suppurative otitis media.

Autopsy Report.—Puncture of third interspace of chest under water shows presence of air in left pleural cavity. On opening chest wall the heart is displaced to right. The left lung is collapsed. There is a large pleural cavity lined by a pyogenic membrane and filled posteriorly by a thick purulent fluid. The anterior border of the lung is adherent to the fifth, sixth, and seventh ribs just external to the junctions of the corresponding cartilages. This draws the lung out into a fold and partially separates the cavity into two divisions. On removing the lung two perforations in the visceral pleura are seen

near the midlateral aspect which communicate with a pulmonary cavity.

Examination of the lung shows that the tracheobronchial glands are enlarged and caseous; the apex of the lung and a small area at the base are atelectatic and show only a few small caseous

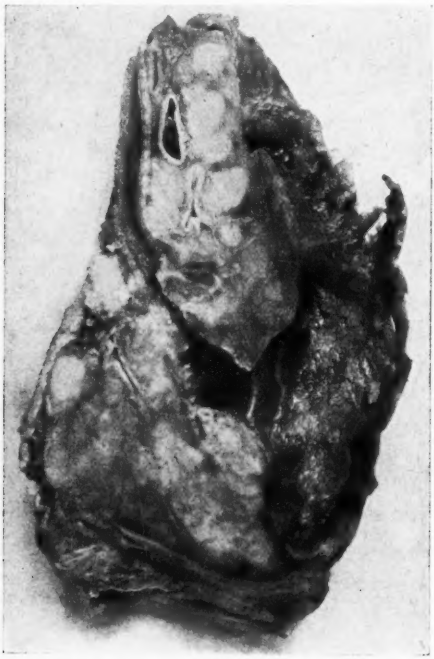

Fig. 329.—Longitudinal section of lung showing caseation and cavity formation. Shows connection of cavity with bronchus and pleural cavity. The adhesions of the lung to the thickened parietal pleura are seen. Tracheobronchial lymph-nodes enlarged and caseous.

areas. The rest of the lung shows advanced tuberculous pneumonia (caseation). In the center of this is a triangular cavity (2 x 3 cm.) with its base at the pleural surface and opening by the two perforations into the pleural cavity. The other end of the cavity communicates with a large bronchus (Fig. 329). The right lung shows scattered small areas of caseation.

The brain shows a thin gelatinous exudate on meninges at base, but no tubercles. There are also scattered tubercles in liver and spleen.

Discussion.—This case shows several interesting features. First the age of the child. Pneumothorax secondary to pulmonary tuberculosis is very uncommon in the first year of life. At this age, when there is involvement of the parenchyma of the lung, generalized tuberculosis usually follows quickly and death takes place before the local lesion has time to spread and break through into the pleural cavity. This may explain the rarity of pneumothorax. As to the diagnosis, the value of the tuberculin test and *x*-ray are obvious; without these the diagnosis would have been impossible when the infant was first seen. By correlation of the clinical findings, the *x*-ray picture, and the postmortem examination it is easy to follow the progress of the lesion. The original lesion in the lung as shown by the *x*-ray in time went on to caseation, then to cavitation, and finally broke through into the pleural cavity. In addition the *x*-ray (Fig. 328) shows very well the pleural adhesions and the thickened parietal pleura—both of which were confirmed by autopsy.

Case II.—Age three months. Was admitted to the Royal Victoria Hospital April 14, 1923, with complaints of vomiting and loss of weight. The family history was negative.

Personal History.—A premature child of 5 pounds, 3 ounces birth weight. Mother did not nurse child. Has been a difficult feeding case since birth, and has suffered from indigestion with attacks of vomiting and diarrhea. Did well for a time when fed breast milk from a wet-nurse. Wet-nursing was discontinued about three weeks ago, and since then has been losing weight steadily. At the age of a week had several furuncles on arm and head. These discharged pus for a time.

Present Condition.—Weight 9 pounds, 1 ounce. Temperature 102° F. Respirations 36. Pulse 152. Patient is a poorly nourished infant. Shows slight cyanosis of skin. Over the occipital region of the scalp there are two furuncles discharging

pus. The surrounding skin is reddened and edematous. Examination otherwise essentially negative. Laboratory findings: Urine acid, specific gravity 1018, trace of albumin, few white blood-corpuscles, no casts. W. B. C., 20,000; R. B. C., 3,800,000; H. B., 70 per cent.

Course in Hospital.—The infant was given a feeding of cow's milk dilution with sugar added, which he took well. On April 14th the boils on head were incised and between 2 and 3 drams of pus were evacuated. Unfortunately cultures of these were not made. On April 15th the temperature was $103\frac{4}{5}°$ F. Another boil was incised and pus evacuated. On April 16th the temperature was 100° F. The patient vomited a small amount of blood and mucus. On the next day the patient's condition seemed somewhat improved. On April 18th the child suddenly collapsed after his bottle, became extremely cyanosed, pulse became very rapid and thready, and respirations labored. Stimulants, including hot water, caffein, brandy, and oxygen, resulted in slight improvement. Examination at time revealed no cause for the attack.

The cyanosis and labored breathing persisted and was relieved somewhat by oxygen given by nasal catheter. Throughout the day the abdominal distention was marked. In late afternoon examination of chest showed limitation of movement of right chest, with diminished breath sounds over this area. The possibility of a foreign body in or a mass pressing upon the right bronchus preventing entry of air was considered. On April 19th the patient's condition was about the same. Examination of chest showed a somewhat hyperresonant note over right chest and distant breath sounds. No coin sound was present. The possibility of pneumothorax was considered, but no definite diagnosis was made. Patient died in afternoon of same day.

Autopsy performed on following day showed multiple abscesses in both lungs. One of these in right lung had ruptured into the pleural cavity, causing a pneumothorax. There was a small collection of purulent fluid in the right pleural cavity.

A smear and culture from the abscesses showed Staphylococcus aureus.

Autopsy Report.—The right lung was collapsed and the pleural cavity contained 50 c.c. of purulent fluid. The right lung adherent posteriorly by fibrinous adhesions. Both lungs contained a number of abscesses varying in size from that of a pea to a cherry. At lateral surface of right lower lobe pus was seen to exude from a cavity about the size of a large cherry. This cavity communicated with the air-passages of the lung. There were no abscesses in the other internal organs. A smear and culture from the abscess showed Staphylococcus pyogenes aureus.

Discussion.—The multiple pulmonary abscesses were probably due to the entrance into the pulmonary circulation of the infective organism. The obvious source of infection was the furunculosis of the scalp. Entrance of the organisms into the circulation would be favored by the low degree of immunity possessed by an athreptic infant. It is unfortunate that cultures were not made from the furuncles, and once again shows the importance of routine cultures of all pus.

Case III.—Age twenty-three months. Was admitted to hospital January 18, 1924, suffering from cough and fever. The family history has no bearing on the case. Patient has been healthy. Had chickenpox at age of three months and pneumonia a year ago.

Present Illness.—On January 9th patient seemed drowsy. The next day she began to cough and was feverish. She had slight nasal discharge. For past five days the cough has been getting more severe and patient has had high fever.

Present Illness.—A small, poorly nourished female child appearing acutely ill. Temperature 102° F. Pulse 150. Respiration 45. The breathing is somewhat labored and is accompanied by dilatation of the alæ nasi. She has a troublesome cough. There is a slight nasal discharge. Lips dry and cracked. No herpes. Tongue heavily coated. No stridor on breathing. Chest is symmetric. No retraction or bulging of soft parts.

The left half of chest shows diminished expansion. The percussion note over right side of chest is hyperresonant. Over the left side the note is almost flat over upper part. It shows an impaired note in axilla and at base. On auscultation there is frank blowing breathing over whole upper lobe of left lung. Over lower lobe the breath sounds are distant vesicular. No

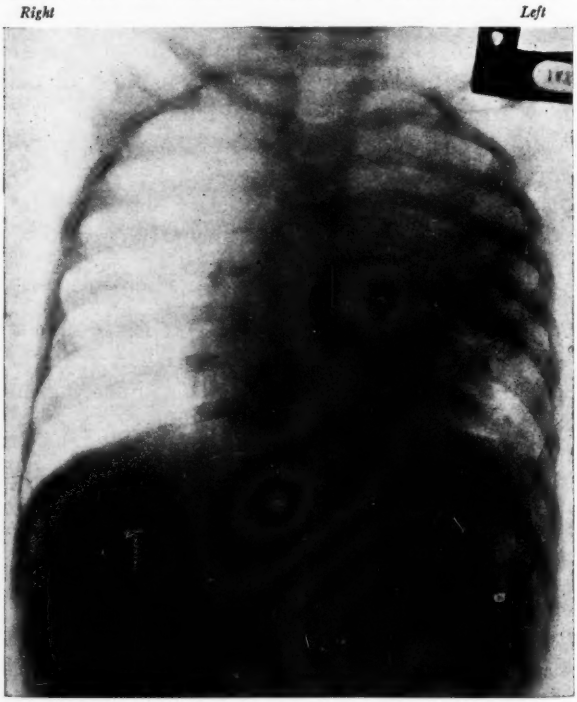

Fig. 330.—Pneumonia of left upper lobe.

râles heard. Over right lung breath sounds are harsh and vesicular. The heart is in normal position and sounds are clear. Abdominal examination negative. Genito-urinary, nervous, and locomotor systems negative.

Laboratory Examination.—W. B. C., 27,600. Intradermal tuberculin test 1/10 mg. negative. Urine 1030, acid, trace of albumin, few granular casts, occasional white blood-corpuscles.

Course of Disease.—*x*-Ray taken on January 19th showed pneumonia of upper left lobe of lung, little if any fluid present (Fig. 330). During the next four days the child's condition became slightly worse. Respiratory distress increased. The dulness at left base became much more marked and the breath sounds very distant. The presence of fluid was suspected. On

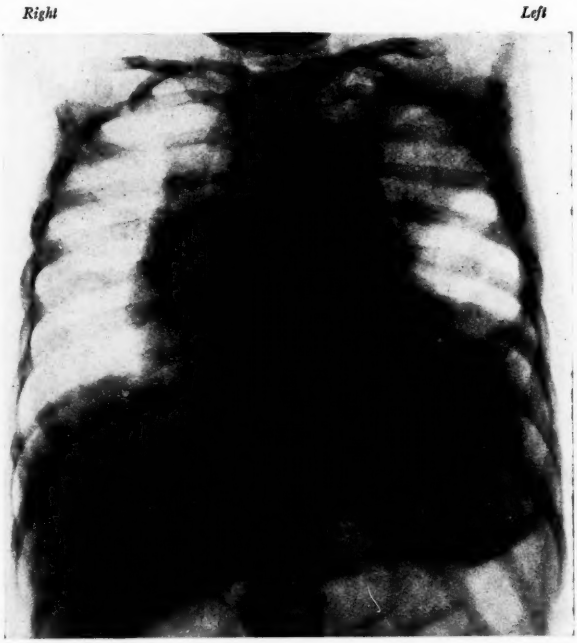

Fig. 331.—Pneumothorax of left pleural cavity. Adhesions at level of eighth rib. Heart and trachea displaced to right.

January 24th the percussion note over the upper part of left lung showed a definite change. The note, instead of being dull, was hyperresonant and tympanitic. Breath sounds were very distant and the coin sound was present. The heart dulness was definitely displaced to the right. The signs suggested pneumothorax. An *x*-ray of chest was taken and the radiologist gave the following report: "The heart and trachea are pushed over

to the right. There is a pneumothorax of the left chest extending from the apex to the base, except for a band of adhesions at the level of the eighth rib. The left diaphragm is invisible, suggesting presence of fluid (Fig. 331). On January 25th fluoroscopic examination showed a movable shadow at left base, showing waves on shaking patient. Definite evidence of fluid.

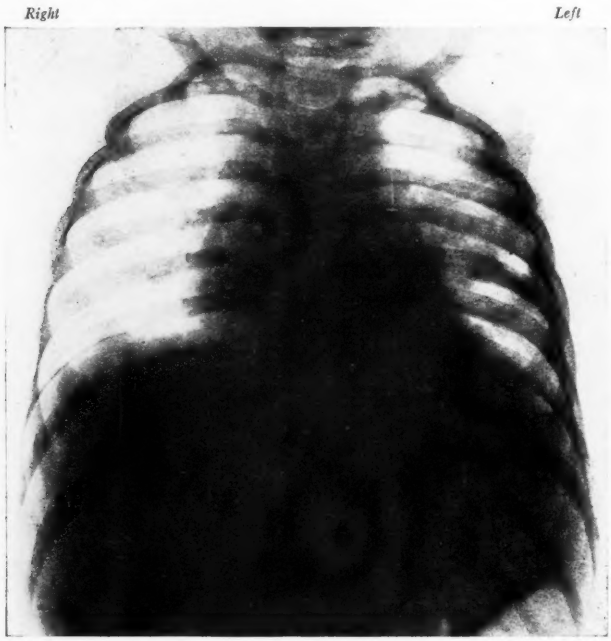

Fig. 332.—Twenty-four days after operation. The lung has expanded almost completely and heart is returning to its normal position.

Inspection of chest showed definite fulness and fixity of left side. Exploratory puncture at left base in ninth interspace was done on January 26th, and about $2\frac{1}{2}$ ounces of creamy pus obtained. Direct smear of pus showed Gram + diplococci, and also cocci in chumps. Culture of pus showed Staphylococcus pyogenes aureus. The patient's condition remained about the same for the next few days. There was considerable dyspnea.

Physical examination and fluoroscopic examination showed steadily increasing amount of fluid in chest. It was decided that operation was advisable.

On February 1st a thoracotomy was performed by Dr. Scrimger at site of puncture wound. The opening was intercostal, and on reaching the pleural cavity there was a rush of creamy pus and air. The dressing was so arranged that a one-way valve was formed, allowing for the drainage of pus, but preventing ingress of air. Following operation the child improved steadily. However, as the discharge continued with slight fever, it was felt that a pocket of pus might remain. On February 21st a rib resection (1 inch) was done (ninth rib). Little or no cavity was found. On February 25th *x*-ray of chest showed that expanison of lung had taken place. No evidence of fluid. Heart returning to normal position (Fig. 332). Examination on March 1st shows the wound has healed. The left side of chest moves fairly well. There is an impaired note at left base. On auscultation breath sounds are well heard in front. At base behind are distant. A second tuberculin test of 1 mg. intradermally has been negative.

Discussion.—Pneumothorax complicating empyema is well recognized. It may be due to erosion and perforation of the visceral pleura from without. More likely causes, however, are the formation of abscesses in the pneumonic lung, with rupture into the pleural cavity, or rupture of an emphysematous area in the affected lung as a result of coughing.

Tuberculosis may be excluded here by the course of the disease and the repeated negative tuberculin reactions.

CLINIC OF A. G. MORPHY

ROYAL VICTORIA HOSPITAL

CONVULSIVE SEIZURES

SURPRISES are in store for anyone who sees many cases of so-called epilepsy unless, of course, he makes up his mind beforehand not to be surprised at any unexpected turn of events in some of his cases, particularly those in which the psychogenetic factor is marked. Even in so-called essential or idiopathic epilepsy there are instances here and there of remissions, and in the psychogenetic cases recovery sometimes occurs unexpectedly and in curious ways.

The following 3 cases are interesting as presenting psychogenetic factors in varying degrees:

Case I.—A. C., aged twenty-four, came to the Neurological Out-door Department at the Royal Victoria Hospital in June, 1919, complaining of peculiar attacks which had begun two and a half years previously.

A peculiar giddy feeling would come over him, an out-door scene would flash before his eyes, and a strange thought which he has never been able to define would course through his mind. His fellow-clerks told him that he would lift up his right arm, saying, "There it goes again, what the devil is it? Well I'll be damned." He would become deathly pale and pinched, and stagger about until he regained consciousness and control.

His mother described attacks at night in which he made a peculiar noise, his eyes were fixed, and he seemed to be in a convulsion, but his limbs did not work. He had never wet the bed or bitten his tongue in attacks.

History.—Born in England, perfectly healthy with exception of a convulsion at six months supposed to be due to

teething. Rather precocious. Very bright at school. Graduated from school at twelve years, average age being fourteen. He had diphtheria at twelve years, pneumonia at sixteen, rheumatic fever at nineteen. In this illness was three months in hospital.

His attacks were infrequent at first, but gradually became more frequent and more severe. Just before coming to the neurological clinic he had lost his position.

Physically he was well developed. His thyroid was very slightly enlarged and there was very slight prominence of the eyes. Systolic murmur was heard over the heart. Neurologic examination negative, Wassermann negative, urine normal.

Mentally he was rather dull and dispirited. His memory was poor. He was worried over loss of his position, and was becoming irritable.

His mother described three kinds of attacks: "Talking" attacks during the day; night attacks in which he wriggles over the bed, rubbing his stomach, quite unconscious; and "noisy" attacks, in which there is a cry followed by a slight convulsion.

Progress.—He was given bromids, but his attacks continued. Peculiar and ridiculous thoughts passed through his mind, and at times he was confused, saying meaningless phrases. His memory was impaired. Recent events made little or no impression on him. He lost track of time; could not tell whether it was morning or afternoon. If told the date, could not recall it after two or three minutes. He would forget whether he had had his meals. He became indifferent as to whether he lived or died. When in company of other people he felt as if he was not one of the crowd. He stated later that he would have committed suicide if he had had the courage.

In March, 1920 bromids were discontinued and he was given luminal. Marked improvement noted. Mental tone brighter, memory improved. One attack per day.

In September, 1920 he stated that his memory for past events, with the exception of a certain period of about four years, had returned.

This memory lapse was remarkable. His marriage in April,

1916, and the birth of his child in 1917 were forgotten. He was questioned repeatedly on this point, but invariably gave the same answer. The incident of moving to another house in 1920 was the end of the period of amnesia.

As his intellectual functions became brighter he became more assertive, talkative, and grandiloquent, using long words by preference. He was affected, sententious, and egotistic. His voice had a wooden quality, with very little alteration of pitch. There were many opportunities of observing him, as he worked in the Industrial School for Epileptics. He was arbitrary, irritable, and intolerant of others. After he had worked for about a year it was necessary to ask him to leave. He was intolerable.

In 1921 he continued to have light attacks. His sexual desire and power, which had gone from him about two years previously, had not returned. His memory blank now covered about two years, many former gaps being now filled.

In April, 1921 he had twelve attacks in eleven days, having fallen to the ground once.

At this time he was taking 4 grains of luminal per day, increased later to 6 grains.

In April, 1922 he reported fewer attacks, and his attendance at the clinic became irregular, and in October, 1922 he came for the last time.

After an interval of two or three months the Social Service followed him up at my request.

The report was that he had had his eyes examined in June, 1922, and got the glasses prescribed. "The effect was marvelous." He had had no more fits from that moment. Stated that he had no anticipation of this, and that there was no autosuggestion about it.

He then began to cut down his luminal gradually, reducing it to nothing in about two months.

His spirits improved. He became less irritable, gradually more cheerful, sexual power returned.

Notes taken February, 1923: His memory for the period of his life above noted is still imperfect. He is jolly, associates with friends, laughs easily. Intellectually he says he is quite

different. He can learn and remember easily. He is studying shorthand. From my own observation I can see that he is a changed man. He is bright, his speech is not slow. He is not so pedantic; is jolly and good-natured, and does not mind talking freely about his former pecularities.

Several months later A. C. telephoned me to say he was quite well, and was working regularly in an office.

How shall we diagnose this case? His attacks, as described by his mother and wife, conformed to our conception of epileptiform attacks in that there was loss of consciousness preceded and followed by automatic movements. On one occasion he fell to the ground. There was no detectable physical abnormality which could be held responsible unless we attribute some influence to a slight enlargement of the thyroid and his healed endocarditis.

If we assume that an unnoticed encephalitis might have accompanied or followed his diphtheria or rheumatic fever, causing a more or less diffuse sclerosis in the brain, with increasing contraction of the scar tissues, how shall we account for his recovery?

If we fall back on psychogenic theories we are nearer to probability. And yet the patient denied all emotional stress preceding the onset of his attacks, with the exception of one incident in which an old school friend had robbed him of watch and money and disappeared. He admitted that this gave him a shock, but it is difficult to believe that this was sufficient.

The mental dulness in the earlier part of his illness may be partly attributed to bromids, but not wholly. The loss of memory for a certain definite period of time suggests an amnesia of psychogenic, not of organic or toxic, origin. His impotence at this time seems to harmonize with a psychogenic amnesia.

At the same time the arbitrary turn of mind, the irritability, the wooden, inflexible voice, the obstinacy, and intolerance of others are characteristics regularly found in essential epilepsy.

It may be remarked here that the suggestion was frequently made to A. C. that his case was not one of true epilepsy. So that when the "marvelous change" came over him with a look through

a pair of glasses that suited him he might have been ready for it. Whether luminal increased his irritability is open to question. A peculiar circumstance was that he resisted every attempt of mine to reduce his doses of luminal at the very time when he was carrying on a systematic reduction himself.

His attacks having disappeared, and his medication stopped, his mentality and disposition changed. His recovery, so far, is complete. In December, 1923 he reported that he was quite well, working in an office. The only possible diagnosis seems to be epileptiform attacks in an unstable individual with marked psychogenetic element.

Case II.—H., Jewess, came to Clinic July, 1921, complaining of "attacks." Age at that time sixteen.

History.—No convulsions in infancy. Mother died of tuberculosis four years ago after prolonged illness, during which time the patient looked after her, did the housework, and went to school. On three occasions had seen father beat mother. Was frightened and ran away.

Father deserted family during mother's illness. After her mother's death H. felt ill and weak.

At fifteen she went to business college. First attack occurred then. States that she remembers nothing about it.

Her description of attacks varied. At one time she would fall to the floor four or five times, picking herself up each time, her legs and arms jerking and teeth chattering. Another time she felt nervous when getting out of bed, and fell while stooping over to put on shoes, but did not lose consciousness. All morning she felt jerky and could not control movements; spilled cup of cocoa over her dress. Severe attack came on later. Remembers lying down and putting cushion under her head before it came, then lost consciousness. Felt better when she woke up. Other attacks were very suggestive of hysteria. She complained of having creepy feelings in her feet, mostly at night, and of feeling "nervous inside." She dreamed frequently of unpleasant things. Sometimes she woke up suddenly in the morning with jerking movements of body, arms, or legs, which were apt to

continue for half an hour or longer, and she could not control them.

She stated that she had been subject to "jerking attacks" for about two years before her "unconscious attacks" came on, and that they started when she was under great strain, going to school and nursing her mother.

The woman in whose house H. lived stated that H. sometimes had attacks in which she fell down, foamed at the mouth, and sometimes bit the tongue.

H. stated that she found that her jerking attacks were more frequent when she was troubled in her mind.

At one time these movements kept on during a whole night, and H. made a snoring noise. On another occasion she jerked so much that she fell out of her chair and hurt herself, and blood came freely from her mouth. She lay on the floor for about half an hour, snoring and rolling over. Her face was flushed and her eyes looked frightened and very shiny. After waking up she did not know what had happened. This account was given me by Miss V., with whom H. lived. Miss V. states that H. is very sensitive and childish, very easily moved to laughter or tears.

I personally witnessed an attack. H. was working at a loom in the school. Her arms began to jerk. She went and lay down. Then she had a typical major epileptic attack.

After this there was no more doubt about attacks with loss of consciousness.

In 1923 attacks of all kinds gradually ceased. In August she confessed, shamefacedly, that she had not been taking her luminal for three months. Since then she has had no attacks and has taken no medicine. A note taken December, 1923, states that H. had been very hysteric lately, laughing and crying immoderately without obvious cause. She admitted that her mother had been very much in her thoughts lately, appearing before her as H. had seen her years ago, looking very ill.

Since then she has reported herself quite well.

This case might be labeled hystero-epilepsy if there is such a thing. The psychogenic element is very prominent. For a

year or more the diagnosis of hysteria seemed the right one. Then came the confirmation of attacks with loss of consciousness. The jerking attacks—what shall we think of them? We may call them hysteric, but does not this word resemble a convenient bag into which to put labels. There is a certain resemblance between a series of jerking attacks followed by a major convulsive seizure and jacksonian attacks spreading by waves until consciousness is lost. In either case there must be loss of cortical control. Shall we call these jerking movements motor auræ? In some instances they lasted hours at a time, and were not followed by loss of consciousness. If we call them hysteric we must assume disturbed function of higher cerebral centers—dissociation, retraction of the field of consciousness, or what you will—with involuntary rhythmic motor discharge, with repressed emotional storms as a basis. And if we admit that a "hysteric" attack can run on into an "epileptic" attack, where shall we draw the line?

As to treatment, luminal was given, whether rightly or wrongly it is hard to judge. H.'s dreams, emotions, and conflicts were investigated as far as time allowed, and much suggestion and encouragement were offered. An incident which apparently had some influence on the course of events was a letter received by H. from her father after an interval of two years, in which he enclosed money and stated that he had begun life anew and intended to do his duty by his children.

On last reports, a few days ago, H. was quite well, although it was evident that her emotional balance was rather fine and apt to topple over one side or the other.

Case III.—C. V., aged twenty-one years, unmarried, came in January, 1924 complaining of attacks of unconsciousness, vomiting, nervousness, and sleeplessness.

Her first attack accurred in December, 1919, and she was unconscious (her own statement) all night. Remained in bed a month with severe headache. A year later she had a similar experience. Since then she has an attack every three or four weeks. Each one is preceded by the feeling of a hot flush all

over her body, most marked in her head and face, then a pins-and-needles sensation. Her heart begins to jump, light and voices fade away, and she knows nothing more. Knows nothing of what has been done for her during the attack. Afterward she feels exhausted and is apt to vomit.

History.—No convulsions in childhood. Used to wake up frightened and scream. Says she used to get up and do funny things in her sleep. Had nocturnal enuresis until nine years of age. Until two years ago her mother had to wake her up on account of her screaming in her sleep. Was always easily frightened and very sensitive. Is still scared of ghosts. If agitated she is liable to vomit.

Family History.—Her eldest sister has been subject to fits since seven years of age. Patient has always been frightened seeing her sister in a fit, fearing sister would die. Two years ago her mother died suddenly, and this gave her a great shock. Two brothers and one sister died of tuberculosis.

Dreams.—Often dreams of her mother's death. Sees her mother sitting up in her coffin and looking quite natural. Often dreams of going to the cemetery and seeing her mother and father lying on the ground. Often dreams of funerals. Waking up in the night after bad dreams she hides under the bedclothes for fear of ghosts. Often dreams of falling from a height, of drowning, or of being pursued. She is sure to dream an unpleasant dream over again if she wakes up in a fright.

Description of a Seizure.—Without warning the patient becomes rigid and remains so for about fifteen minutes, seemingly unconscious. Her hands are clinched, arms and legs outstretched and rigid, toes plantar flexed. Eyelids closed, eyes rolled up and to right, pupils medium size, reacting to light. Skin appeared blotchy, with red and white patches. Kept moaning, "Oh, my." Pulse 192 during the spell.

Physical examination shows a well-developed, healthy looking young woman, with no evidence of somatic disease. There is, however, some tenderness in the right lower quadrant of her abdomen. Her reflexes are normal. No disturbances of sensation. Wassermann negative.

In this case we diagnose hysteria. But there are certain features in the attacks which resemble those seen in an epileptic paroxysm. There is a gradual reduction of consciousness with sensory auræ, namely, paresthesiæ, then the stage of decerebrate rigidity, with further lowering of consciousness, in which sensation and perception fade away; the feeling of exhaustion as consciousness becomes integrated, followed by headache and vomiting. Closer observation may yet reveal a clonic stage exhibited as restlessness after subsidence of the rigidity. In our tendency to attribute a certain amount of simulation or deception to hysteric attack we are prone to disbelieve the patient's affirmation of complete unconsciousness of the whole procedure. But are we justified in so doing? There is no question that through emotional disturbances control of the higher cerebral centers is cut off, and an unregulated stream of motor impulses from lower centers innervates the muscles, just as in an epileptic fit.

A brief summary of these 3 cases, then, would be the following:

Case I. Epileptiform attacks with momentary unconsciousness, automatism and increasing mental deterioration and irritability, followed by unexpected clearing up, cessation of attacks, and resumption of former occupation.

Case II. Attacks seemingly hysteric in a girl of poor heredity and unstable make-up following life tragedies, with occasional typical epileptic paroxysms, and symptomatic recovery.

Case III. Hysteric attacks in a girl with very poor heredity and environment, superstitious, subjected to very severe stress, these attacks resembling in some degree epileptiform attacks.

Rosett's theory of normal epileptoid reactions bears upon all cases of convulsive seizures. Muscular contraction, he says, even when initiated voluntarily implies a reduction of consciousness in proportion to its intensity, and it therefore follows that narrowing of the field of consciousness from any cause, such as sudden shock or emotional stimuli, should result in muscular activity.

Also the normal reactions to such stimuli resemble epi-

leptic paroxysms. A rapid contraction of the field of consciousness from exposure to sudden and painful stimulus, with paresthesiæ, such as vertigo and epigastric sensations, during the gradual disintegration of consciousness, and automatic actions; then the stage of tonic rigidity, "scared stiff"; then clonic movements and desire to urinate, followed by stage of exhaustion.

In all 3 cases we have seen convulsive seizures coming on as reactions to painful emotional stimuli, these being not so plainly made out in the first case as in the second and third; the first one labeled epilepsy; the second, hystero-epilepsy, and the third, hysteria.

It seems advisable to pay more attention to mechanism and not allow our conceptions of cases to be influenced by our tendency to label them with names of diseases.

Cases I and II offer much encouragement to persist in a hopeful attitude toward all such cases.

CLINIC OF DR. H. B. CUSHING

ALEXANDRA HOSPITAL FOR CONTAGIOUS DISEASES

HEMORRHAGIC DIPHTHERIA

GENTLEMEN, our patient today is a little French Canadian girl of eight years who understands no English, so cannot take in our remarks. On examination she is obviously suffering from diphtheria and, more than that, is evidently not going to recover from the disease. My reasons for these two statements will be evident shortly. Now of all the infectious diseases diphtheria is the best understood and the most under our control. With the knowledge at our disposal it is possible to stamp out entirely the disease in civilized communities, absolutely to protect any child against contracting it, and, if contracted, we have a specific which should certainly cure every case treated in time. The history of this case then should contain some explanation of why she is dying of the disease in the midst of a great city.

First, as to her past history: She comes of a healthy family, both parents are alive and well, and she has a younger brother and sister in good health. She herself was a normal healthy child until she went to school at the age of five years. In the three years that have elapsed since that time she has suffered from measles, whooping-cough, scarlet fever, chickenpox, and mumps in succession, and finally diphtheria. Surely something is wrong in the hygiene of our schools that such a price should be paid for an elementary education. However, to keep to the subject in hand, it is obvious that her resistance to disease must have been impaired from her multiple infections. As to the diphtheria, we can find no history of exposure to the disease on questioning the parents or inquiring from the school, although

we know she must have acquired it from some other case within two weeks. This failure to obtain a history of infection is more or less characteristic of this disease. In only about 10 per cent. of our diphtheria cases in this hospital can the infection be traced. The usual explanation given is that the infection is due to mild unsuspected cases or to healthy carriers who are undetected. Consequently, the importance of immunizing all children against the disease, which can readily be done by the use of the toxin-antitoxin method. I need not say this has not been used in this case.

The present illness began eight days ago. She was noticed by her parents to be feverish and to have lost her appetite and complained of a sore throat. She was kept at home from school and stayed in bed part of the time, but was not thought to be seriously ill, and no doctor was consulted, as she was considered to be suffering only from a cold. This insidious onset of diphtheria with mild fever and little general constitutional disturbance explains why so many cases are not treated in time. Three days later three other symptoms were noticed: there was a foul odor to her breath, she had a nasal discharge, and her neck was considerably swollen at the sides. This would have made the diagnosis obvious to any physician, but, unfortunately, the parents, on the strength of past experience, decided the child had mumps, and, being in poor circumstances, did not call the doctor. This was, really, the turning-point of the child's illness. If diphtheria is recognized and actively treated in the first three days recovery should follow. After this the toxins already absorbed are beginning to manifest their results, and although the disease may be arrested by sufficient doses of antitoxin, the effects on the various organs cannot be prevented and the outlook is doubtful.

Two days later, as the patient was not improving, a physician was called in. The extensive false membrane covering the throat made the diagnosis clear, and 2000 units of antitoxin were administered subcutaneously; the same afternoon, the child's condition not being improved, she was sent in to this hospital. Under the circumstances the use of this dosage of

serum suggests a man who, discovering a house in flames, stops to get a tea cup of water to throw on the blaze before summoning the fire department.

On admission to the hospital the patient was found to have the cervical glands greatly swollen, to have a "bull neck," in hospital slang, always a sign of serious import. She had false membranes covering the entire pharynx and soft palate, with considerable edema of the throat. There was a sanguinopurulent discharge from both nostrils, showing the nasal cavities were involved, and she had a croupy cough and hoarse voice, showing the larynx was involved also. She had rather an ashen pallor, and the pulse, while regular and full, was distinctly low in tension; the urine showed a small amount of albumin and a few granular casts. There were, then, distinct signs that the circulatory system and kidneys were already affected by the toxins of the disease. The first consideration was the administration of serum. The dose of antitoxin should be regulated, first, by the time the disease has existed: second, by the extent of the membrane; third, by the virulence of the process so far as it can be estimated, and fourth, by the region involved. In this case everything pointed to the use of a maximum dose; the patient had been sick seven days, the membrane was most extensive, the edema and glandular involvement showed a virulent infection, and the larynx was involved. Again, if antitoxin is given subcutaneously a comparatively small amount is found in the blood within twenty-four hours, and it is not until three full days that it reaches its full concentration in the blood as shown by actual experiment. Therefore in a desperate case like this if any beneficial effect is to be obtained the serum should be used in large doses and intravenously. This little girl was given 10,000 units intravenously and 15,000 more intramuscularly to maintain a continued effect.

The temperature, which was 100° F. on admission, rose in two hours to 102° F., the usual reaction to antitoxin, and then rapidly sank to normal. Today, twenty-four hours after admission, the temperature is 97° F., the pulse 70, the swelling of the neck has greatly diminished, the nasal discharge has

practically ceased, and the membrane in the throat has become thickened and sharply limited, and is beginning to separate round the edges. Anyone without much experience in these cases would think the patient on the high road to recovery, but one must remember that an immense dose of toxin must have been absorbed in the first seven days of the illness; that this cannot be neutralized by any amount of antitoxin given subsequently, as it is already fixed in the tissues and will show its effects after a week or ten days or even longer in the case of the nervous system. Again, a fresh set of symptoms has developed today, and it is these symptoms which I wish specially to discuss. The disease has taken on a decided hemorrhagic tendency. There has been oozing of blood from the nose and throat, there are petechial spots round the elbows, shoulders, knees, and buttocks; wherever a hypodermic has been given or there has been a slight contusion there is a distinct ecchymosis; one can find at least a half-dozen of these subcutaneous ecchymoses in various parts.

Now the occasional occurrence of this hemorrhagic tendency has been noted in all the acute infections, and it is always regarded as of serious import; one need only refer to "black" smallpox and "black" measles as examples. However, in none of the infections is the hemorrhagic form a more definite entity or of more ominous significance than in diphtheria. Of all the cases that have occurred in the Alexandra Hospital during the past ten years, no clearly marked case has recovered or lived more than three or four days from the onset of the hemorrhagic manifestations. They all died not of loss of blood, but of progressive toxemia and cardiac failure. The pessimistic prognosis given in this case at the beginning of the clinic is then explained.

In recent cases in this hospital an attempt has been made to explain the condition by a careful examination of the blood in all cases which either showed the condition or which, by the general symptoms and toxemia on admission, were thought likely to become hemorrhagic. I might state here that the hemorrhagic condition only develops in cases with extensive

membrane and severe toxemia, and those who show by marked edema and glandular involvement a virulent infection. I never saw it occur in a case treated by antitoxin during the first three days of the disease, and it always develops during the second week of the disease, usually, as in this case, about the eighth day.

The blood examination in this little patient gave the following findings: red blood cells, 4,850,000; hemoglobin, 85 per cent.; leukocytes, 16,000 yesterday and 22,000 today; blood-clotting time five minutes yesterday and four minutes today; bleeding time, eight minutes yesterday, thirteen minutes today; blood-platelets, 225,000 yesterday, 315,000 today; stained smears show red cells practically normal, and the increase in leukocytes to be due to an increase of polynuclears. Let us see what we can learn from these findings. In the first place, in spite of the marked pallor there is practically no anemia. Certain text-books state that the toxemia of diphtheria causes an early and very marked anemia, but I have never found this to be true, although I have examined many cases; the pallor, which is so characteristic, is due to circulatory failure or vasomotor disturbance, but not an actual anemia. Second, there is a noticeable and increasing leukocytosis; this is not found in early cases of diphtheria, but is always present in severe and late cases, and is of serious prognosis if very marked and without any secondary infection to explain it. But the main interest in our examinations is to try to establish some cause for the hemorrhagic tendency. The change causing the bleeding must be either in the hemopoietic system, the blood itself, or the blood-vessels. As to the first, we find no evidence; the cells are apparently normal and there is increase in leukocytes and platelets, apparently the blood-forming organs are functioning. As to the blood itself, there is no evidence of hemolysis, the number of platelets is not diminished, and, most important of all, the clotting time is normal or even shortened. Therefore we are forced to the conclusion that the change must be in the endothelial lining of the capillaries, which is presumably affected by the severe toxemia and causing the increased bleed-

ing time and tendency to spontaneous hemorrhages. The suggestion has been made that the hemorrhages may be due to a secondary infection with some hemolyzing organism, but, in the first place, there is no evidence of hemolysis, and in the second, blood-cultures are almost uniformly negative.

As to the treatment of the condition, the uniformly fatal outcome makes this hopeless. Death, as I have said, occurs from cardiac failure, not from hemorrhages, and the hemorrhages are only an outward sign of the intense toxemia. The only treatment is prophylactic and has already been outlined. The immunization of the children against diphtheria, the endeavors to administer antitoxin early, in sufficient doses, and in a suitable manner should lessen the perecntage of these deplorable cases, which, while not very common, are still no rarity in any infectious service.

Subsequent note: The patient described above became progressively weaker, more hemorrhages occurred in the skin, there was slight oozing of blood from all the body orifices, and the next day marked signs of circulatory failure were manifest. Vomiting began, as is usual in all these cases of cardiac dilatation after diphtheria, and the child died somewhat suddenly on the tenth day after the onset of the disease. An autopsy showed remains of a false membrane lining the whole pharynx and extending down the trachea as far as its bifurcation. There were small hemorrhages scattered through the substance of all the internal organs, with a larger one in the left perirenal tissues, which involved and practically destroyed the left suprarenal gland. There was early degenerative nephritis and myocarditis.

CLINIC OF DR. W. F. HAMILTON

ROYAL VICTORIA HOSPITAL

DIABETES INSIPIDUS

THE following cases illustrate a clinical syndrome manifestly induced by a variety of causes which are gradually becoming less obscure as knowledge of anatomy and physiology increases.

Case I.—The first patient is a male aged nineteen, born in Canada, of Hebrew parents. He was admitted to the Royal Victoria Hospital on March 19, 1924, complaining of thirst, of passing large quantities of pale urine, and needing to do so very frequently both by day and by night.

Family History.—His father died at the age of fifty-six, the cause unknown to the patient. His mother died in 1918 at the age of forty-eight of influenza. An infant brother died of an unknown cause. The other members of the family, six in number, are alive and well. There is no family history of diabetes or of tuberculosis.

Previous History.—At the age of two the patient fell against a fire fender, cutting the skin over the left temple, where a small scar is now to be seen. As this is stated to have caused but little or no trouble at the time, it would appear as if this injury was quite insignificant in the case. Of children's diseases the patient seems to have had attacks of almost if not quite all usual in early life. At twelve the adenoids and tonsils were removed. His habits are good. He is employed as a clerk in the C. P. Railway Offices in Regina, where he has lived for two and a half years.

Present Illness.—The patient states that he was well up to 1919, when, while attending school, he noticed that fairly suddenly he became excessively thirsty, and that he needed to ask

his teacher's permission to go for a drink. The thirst is more marked now than at first. He needs from 3 to 8 glasses of water per hour besides a quart of milk per day—about 20 to 25 pints of fluid in twenty-four hours.

The urine is passed about every hour during the day and about every two hours by night. Slight burning on micturition has been noticed on several occasions. A tingling sensation has been felt in the left hand from time to time. He has kept on with his work and seems so far to have suffered only inconvenience on account of his complaints. Headache, fatigue, loss of appetite, or failure of vision have not been experienced.

Present Condition.—The patient is a youth of average height and weight, with normal temperature and pulse-rate, well nourished, of good color, and with abundant hair. The thyroid and lymphatic glands are not enlarged. The hands are rather oddly shaped, the middle and fourth fingers diverging somewhat from the midphalangeal joint outward; the terminal phalanges appear very small. The digestive system is practically negative—a few teeth showing slight caries. Examination of the respiratory system is also quite negative. On admission the blood-pressure was rather high considering the age of the patient. The subsequent fall to 130/70, with normal physical signs throughout the cardiovascular system, including an electrocardiogram, led us to attach but little importance to this observation.

The urine, on admission, was voided about every two hours. It was very pale—neutral, specific gravity 1004, amount 11,000 c.c. (Fig. 333). A faint trace of albumin was found from time to time, but no casts. The phenolsulphonephthalein output at the end of two hours was 57 per cent. The blood urea 0.84 gm. per liter; the urea concentration factor 39.

Blood-count: Red blood-cells, 5,200,000; white blood-cells, 5900; hemoglobin 85 per cent., with a normal differential white cell count.

The Wassermann test, both in blood and cerebrospinal fluid, was negative.

The Nervous System.—The patient is rather restless and excitable; very anxious to be cured. The sensation and motor

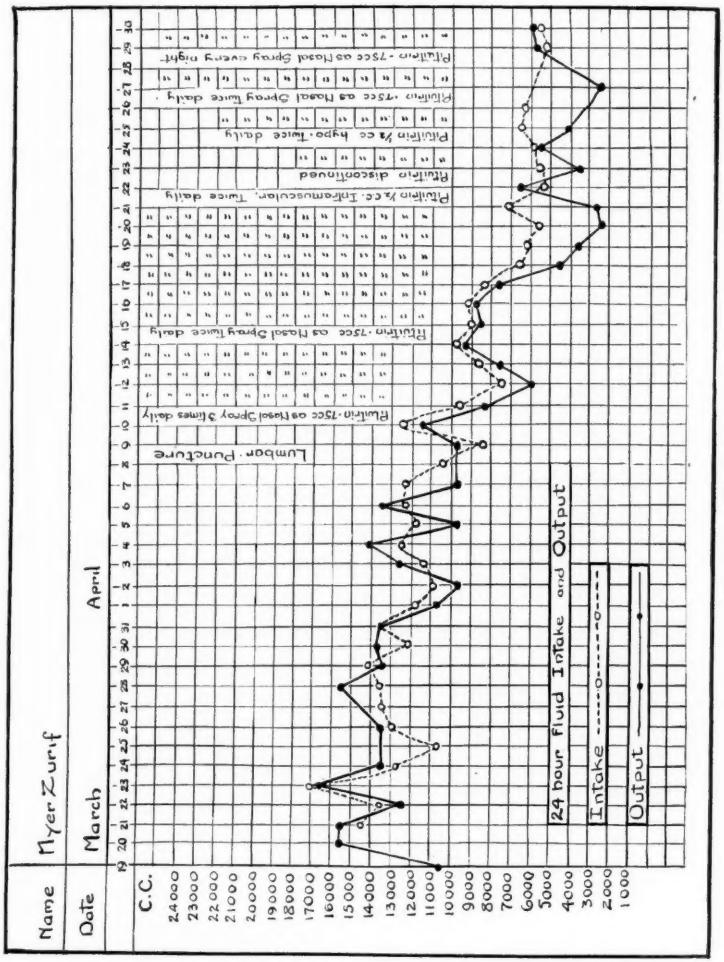

Fig. 333.—Chart of Case I.

functions all normal. The reflexes are not exaggerated. Eyes are normal in every respect, including visual fields and fundi.

A radiogram of the skull shows the sella turcica rather small, clearly outlined, and almost completely roofed over.

Lumbar puncture yielded a few cubic centimeters of clear fluid not under increased pressure and containing but 5 cells to the cubic millimeter.

The course of the case, so far as concerns the fluid intake and the output in the form of urine, is clearly seen upon the chart (Fig. 333). By this one may see also how, even before lumbar puncture or any special treatment was tried, the curves show a decided decline. After lumbar puncture and pituitrin this decline is marked; one sees also the signs of effective treatment by a small dose of pituitrin, 0.75 c.c., given by the nasal spray.

The general condition of the patient was considerably improved as shown by an increase in weight, less nervousness, and by a more hopeful outlook.

After a review of 2 other cases we will discuss them together.

Case II.—A clergyman, admitted in 1916, aged forty-six, with the usual complaints of thirst and polyuria, had been in health until nine months before admission, when, without overwork or worry or headache, he developed the condition for which he sought advice.

While in the hospital the urine was greatly increased, reaching as high as 128 to 130 ounces in twenty-four hours. The total output had been much higher before admission. Radiograms of the skull were negative. The blood showed no accumulation of non-protein nitrogen. The patient had already been treated by pituitrin, with much, though temporary, relief. One cubic centimeter of pituitrin, posterior lobe, taken in the forenoon, would enable him to carry through the Sunday morning service, while the same dose late in the afternoon made it possible for him to preach comfortably in the evening.

This patient was advised to continue the use of pituitrin, to take sparingly of protein diet, to seek relief from his parochial duties, and to report later. Several years have passed, however, without any report.

Case III.—Protracted history—great emaciation—late signs of cerebral tumor—death—autopsy.

A girl of eight years came under observation in October, 1916, complaining of thirst and frequency in passing urine. She showed signs of loss of weight. The ordinary infections of childhood and an injury of minor importance to her right shoulder at the age of seven were all that marked a departure from health in her history up to a few weeks before admission. The patient was one of 3 children who were of average health and intelligence. This case showed the typical characteristic of diabetes insipidus—the thirst—greatly increased output of urine of low specific gravity free from both sugar and albumin. The daily output was upward of 4000 c.c.

The phenolsulphonephthalein test gave an unusually high index—96 per cent.—the fundi were normal; and the heart showed a sinus arhythmia.

After a few weeks the patient was discharged unimproved. About three years later, however, she returned (January, 1919). Now the complaints of frontal headache and occasional vomiting were added to those already mentioned when first in the hospital. It was plain that the child's development was retarded. She was drowsy and irritable by turns.

On account of the new phase which this case recently took on a systematic attempt was made to explain the patient's complaints. The eye-grounds were frequently examined, but were always found normal. The blood of the mother as well as that of the father was negative to Wassermann tests. The history of the father included a luetic infection twenty years before, with two years of mercurial treatment. The patient's blood gave a negative Wassermann. The cerebrospinal fluid, although it showed a reduction in the luetic zone with the colloidal gold test and a cell count of 77, was also negative as to Wassermann test. Radiograms of the skull indicated an early closure of the sutures, with rather long and perpendicular posterior clinoid processes. Headaches, drowsiness, hunger, thirst, an unstable nervous state, "hysteric" attacks, irrational, delirious, unconscious epileptiform seizures, periods of mental brightness

and interestedness, occasional vomiting, are terms recorded by the nurse in charge describing her condition over a long period. Increasing weakness, slowness and irregularity of the pulse, subnormal temperature, left-sided ptosis, twitching of the right leg and arm, stupor, and coma preceded death on July 20, 1920, nearly four years after she was first admitted.

The case was regarded as that of typical diabetes insipidus. There seems no reason, even in the light of autopsy, to change one's mind, for there appears but little reason to regard as other than cases of diabetes insipidus those cases in which an organic lesion is discovered. The response to pituitrin was identical with that recorded frequently elsewhere. The anterior lobe was tried by the mouth for a period of three days; then the whole gland was given in the same manner. No change was observed at any time in the urinary output. The chart exhibited shows the intake and output for three periods. For the first period no pituitrin is administered; then for a period of three days $\frac{1}{2}$ c.c. of pituitrin, posterior lobe and pars intermedia, was given at 9 A. M. daily, and was followed by a corresponding period during which no remedy was used. The response was striking, reducing the urine from 3000 c.c. before to 450 c.c. during the administration. Thirst was controlled; the output was lessened, and concentration of the urine was greatly increased.

In these 3 cases we may see the distinctive features of diabetes insipidus. We may see in the tracings also how the urine output is controlled by pituitrin, and in one of them the anatomic condition, later described, is peculiarly instructive.

Diabetes insipidus is a condition but rarely met with in hospital practice. Johns Hopkins Hospital reports for seventeen years show but 8 cases, or 0.007 per cent. In the Mayo Clinic there were 113 cases among 800,000 patients. In the Montreal General Hospital Rabinovitch states that there was but one case in 50,000 admissions.

The 3 cases herewith discussed were found among 62,000 admissions to the Royal Victoria Hospital.

In considering the cause of this rare symptom-complex one's thought centers on the pituitary body, since preparations

from the posterior part are so effectual in controlling both the thirst and the amount of urine excreted. Motzfeldt has expressed a positive view on this point, remarking in his article published in 1918, that during the past four years, or since attention has been directed to the pituitary body as concerned in this disease, there has not been reported a single autopsy finding in which the gland has not been in some way involved.

Within the last two years encephalitis has been followed by the development of the syndrome to which the term is given. One such case is reported by Signorelli (Arch. Pathol. and Clin. Med., Bologna, 1923; Endocrinology, p. 148, January, 1924) and another by Hall (Amer. Jour. Med. Sci., April, 1923).

In view of the wide-spread incidence of encephalitis and the variety of its sequelæ this rare result is not surprising. Basal meningitis, tumor, syphilis, and trauma are also to be included in considering the possible causes.

Through experimental work prompted, no doubt, by clinical observations and pathologic specimens, another view-point of the cause of polyuria, or diabetes insipidus, is urgently demanding consideration. Important contributions to this subject from the experimental side have been made by several workers. The articles by Camus and Roussy (Endocrinology, December, 1920, and by Houssay, 1918) are particularly interesting. This work was done in greater part before the war. After operating on dogs, and noting the results, these workers conclude "it is neither the injury to nor removal of the pituitary body which leads to polyuria in the dog, but rather a superficial lesion at the base of the brain. Total removal of the pituitary body without injury to the base of the brain does not cause polyuria." It would appear that a superficial lesion at the base of the brain is enough to produce polyuria in the animal operated on. The optopeduncular region is that within which injury is followed by polyuria. It lies at the level of the gray substance of the tuber cinereum in the vicinity of the infundibulum. One such experiment induced polyuria for seven months. This article also shows that the polyuria is produced independently of the thirst.

The report of the postmortem examinations in our last case

shows that at the base of the brain a mass occupies the place of all the structures within the "circle of Willis." The tumor obscures the "circle of Willis" and also displaces the optic chiasm and the second, third, and fourth nerves. Attached to it, yet independent of it, is a large pituitary. The corpus striatum and the optic thalamus were pushed to one side. The situation of the tumor showed that it undoubtedly involved that region above mentioned, injury of which produces polyuria.

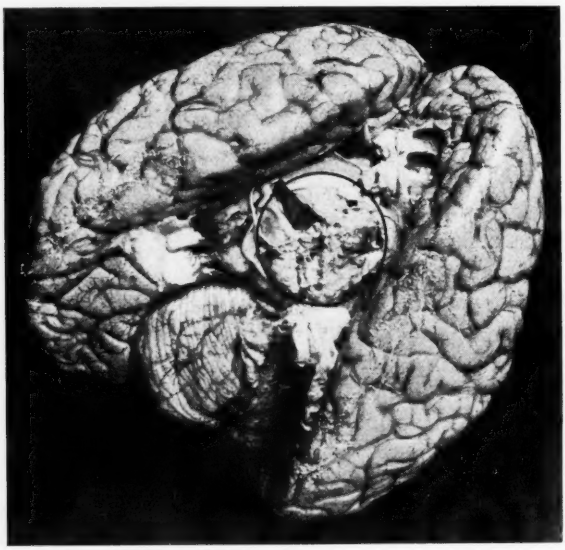

Fig. 334.—Case III. Sarcoma of base of brain. Circle marks the site of the tumor.

Histologically, the anterior portion of the pituitary was involved with new cell growth. When the clinical history of this patient is reviewed there seems to have been a long period of polyuria before the development of any signs which one could ascribe to intracranial tumor. Symptoms of diabetes insipidus were marked in 1916. Late in 1918 headache was first complained of, the first definite suggestion of increased intracranial pressure or of meningeal involvement.

The interest in such cases deepens with more clinical and experimental evidence, which one must admit is not all in favor of the view that diabetes insipidus is a hypophyseal syndrome.

The symptoms of diabetes insipidus are usually pronounced, as illustrated in this patient, as well as in those whose case reports are reviewed. The thirst is intense and urination is frequent, mainly due, it would appear, to the greatly increased quantity of urine secreted. While in twenty-four hours the

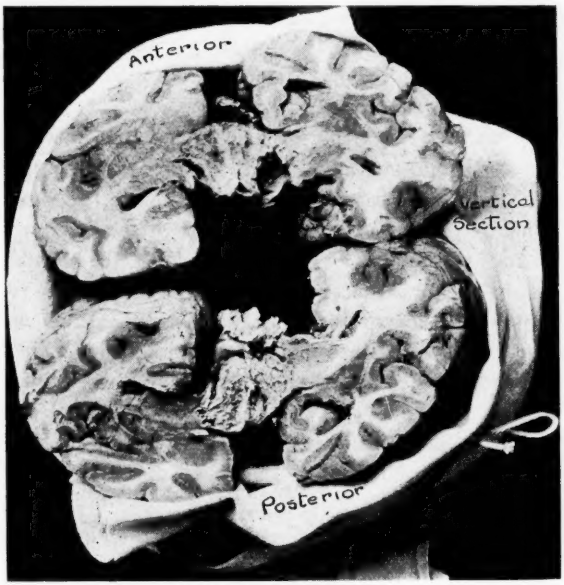

Fig. 335.—Case III.

amount may rise to 8, 10, or 20 liters, Trousseau reported a case in which 43 liters were excreted in one day, the highest on record.

The sequence of symptoms is varied, as the evidence is rather difficult to get. The onset is generally slow. Where it is otherwise, injury or fright seems to precipitate thirst, followed by an increase in the frequency as well as in the quantity of urine voided.

The diagnosis of this condition under discussion may be somewhat difficult. In mild cases one may be in doubt, but in a pronounced type there is rarely any difficulty. Intermittently an hysteric person may void large quantities of urine of low specific gravity, and even thirst may be experienced. These abnormal symptoms are brief or transient. In a prostatic case with hypertrophy similar observations are occasionally made. The relief, however, given by drainage is attended by changes in the quantity and character of the urine. Chronic nephritic cases may suggest diabetes insipidus, but one can scarcely confuse these two conditions once having made a routine examination.

In considering the prognosis one may say this syndrome is not incompatible with long life. In those cases with an organic lesion the outlook is serious. When lumbar puncture does not aid, and in the presence of a luetic history followed by antiluetic treatment improvement is not noticed, the outlook is unfavorable, though life is not threatened. Emaciation and restlessness are ominous. The treatment of this condition is always a matter for serious consideration. It is true the patients may contrive to "carry on," but the insatiable thirst and the frequency of urination interfere alike with the day's work and the night's sleep, thereby inducing nervousness and debility.

To lessen the intake of water in a degree lessens the output, but experience has shown that it is not advisable to push this measure too far, as in so doing the nervous symptoms may become greatly intensified.

Since the kidney is not able to concentrate in an efficient degree without pituitrin, it would appear reasonable to prescribe a diet low in protein and salt.

Among the drugs, from the use of which some measure of relief has been obtained, valerian is highly recommended in the form of ammoniated tincture of valerianate of zinc. In certain cases good results have followed lumbar puncture, but the number of these reported is small. When, however, such salutary results are found, one believes it highly probable that pressure over the optopeduncular region, already referred to, is relieved by draining off the cerebrospinal fluid. The lumbar

puncture should be tried in each case, as improvement has been marked in several instances. Antiluetic treatment may serve a good purpose in a few cases. After all, since Farini in 1913 published his results on pituitrin, however, the problem is rarely solved without recourse to this preparation, the use of

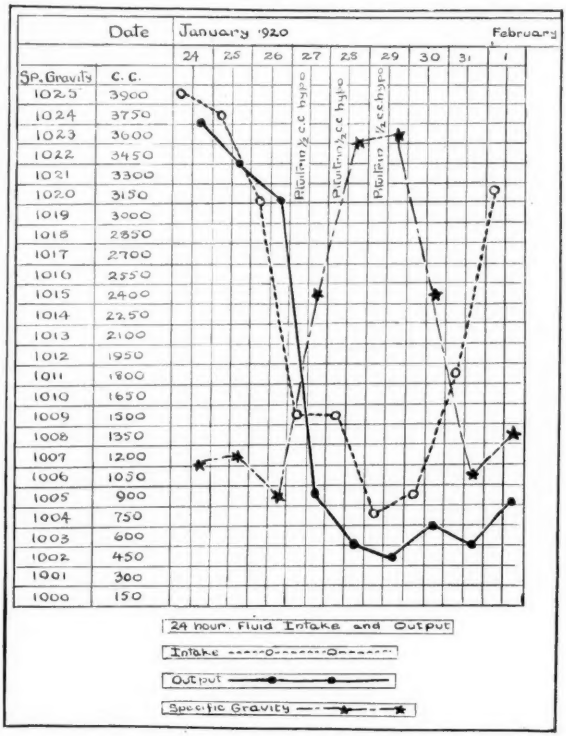

Fig. 336.—Chart of Case III.

which as recommended is expensive and requires care. The best results are secured by the hypodermic method—$\frac{1}{2}$ to 1 c.c. t. i. d.

Attempts have been made to procure results by oral administrations of the fresh gland and also by the use of desiccated

post. pituitary enclosed in salol capsules, 0.75 c.c., 4 h., but these have usually been followed by indifferent results.

The intranasal method is often effective by instillation, spray (Fig. 336), or by using small pieces of cotton moistened with the solution. This may be combined with the hypodermic method for the sake of convenience.

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